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THE
LARYNGOSCOPE.

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RHINOLOGY IN CHILDREN; RESUME OF AND COMMENTS ON THE LITERATURE FOR 1951.

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Toronto, Canada.

This resumé is the product of three members of the Otolaryngological Staff of the Hospital for Sick Children, Toronto. It covers the *Journal of the American Medical Association*, the *Canadian Medical Association Journal*, the *British Medical Journal*, the *Lancet*, the four main British and American otolaryngological journals, and six pediatric journals. The articles have been roughly classified under the headings previously used, and follow almost the same order.

GENERAL ARTICLES ON RHINOLOGY.

Kempton[1] discusses a very common picture of general ill-health associated with chronic or relapsing respiratory infection and having its greatest incidence in the first years of school life. General debility and fatigue, recurring colds, and recurrent or persistent cough are common symptoms along with various other manifestations of minor or borderline ill-health — pallor, poor or fickle appetite with failure to gain weight, indigestion and bilious attacks, and interference with sleep. The picture varies greatly in detail and degree. He notes that Eustace Smith, in 1899, described these children as suffering from "mucous disease"; that G. F. Still thought

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the cause was infected adenoids, and this is still believed by many people; and that Sir Robert Hutchison did not altogether accept either of these conceptions but emphasized the importance of considering the whole child and his surroundings, and especially his diet.

He states that it is when infection settles in a mild chronic form in one or more of the nasal sinuses that the picture of persistent ill-health may gradually emerge. Cold follows cold with quite abnormal frequency — "He's no sooner rid of one than he starts another." Cough is probably the commonest presenting symptom. One may presume that mucopus in the nasopharynx is the commonest cause of cough in childhood. The cough stops when the nasal infection has cleared. Also, iodized oil of about the same viscosity as mucopus placed in the nose at night can be radiologically demonstrated in the lung fields next day.

The full picture of ill-health may develop slowly or rapidly.

He states that examination and investigation are simple procedures. . . . In the great majority of cases no radiological or other hospital investigation is required in the first instance. . . . It is unusual for a radiograph of the sinuses to give evidence of infection which is not apparent on careful examination of the upper respiratory tract by ordinary methods and with ordinary equipment, and a radiologically opaque antrum may sometimes be shown not to be infected. Many of these children do become chronic hospital outpatients. Ultimately recovery to normal health in the filling-out stage of growth which precedes puberty is the general rule, but in many cases there is slight impairment of health in adolescence.

There is probably no group of cases in which treatment as generally carried out is so irrational and ineffective and he cites instances of such.

Rational treatment of this very common picture presents a problem in cooperation and should have three main aims: rest, nasal treatment and exercises. These he discusses in some detail.

Sinus infection usually clears up on treatment with 1 per cent ephedrine and 1 per cent silver proteinate B.P. in saline used as nose drops two or three times daily for a period of about six weeks. Treatment must be carried out regularly with the head held vertically downwards and backwards, and he provides a photograph illustrating the technique. He states that it is all-important that the three main aims in treatment are followed simultaneously. When such combined treatment is possible, a return to full normal health can usually be expected in three to four months.

[*Comment:* In the opinion of the authors of this resumé, examination and investigation of such a patient are not simple procedures and can only be achieved by an otolaryngologist of much experience and by one who has special aptitude in the handling of little children. It requires a patience, a gentleness and an understanding which many eminent specialists do not possess and in many instances do not care to develop. The examination is very difficult and requires the expert use of the nasopharyngoscope, and it must be done in such a way that the child will submit to whatever subsequent treatment is required. The radiologically opaque antrum must not be allowed to remain opaque. Diagnosis is the essential key to proper therapy. Treatment must be rational. At the Hospital for Sick Children, Toronto, large framed photographs illustrate how treatment should be done. The position of the head as shown in the *British Medical Journal* article is wrong. In that position, drops put into the nose reach the cribriform plate and if strong enough may cause alarming symptoms. The treatment should be directed to opening the middle meatus and requires the lateral head low position.]

Andrewes, Lovelock and Sommerville,[2] from the common cold research unit at Salisbury, England, report an experiment designed to determine the effective routes of person-to-person transmission of colds. The original objective was not attained, but other interesting and unexpected findings make the attempt worth recording.

Twelve volunteers landed on an island on the north coast of Sutherland, which has been uninhabited for 12 years. With

them were landed equipment stores and all necessities so that no personal contact between them and the mainland should be necessary for about three months. In the preliminary isolation period of 10 weeks, colds occurred in five individuals, which rapidly cleared up and there were no more during the isolation period. Then the party separated into three groups, A, B and C, which thereafter lived and cooked in separate buildings, and the island was divided into three territories, to one of which each party was restricted.

On Sept. 19, two of the authors landed and joined Party C to see whether normal cold-free people could carry cold virus in their respiratory tracts or elsewhere on their persons and so convey infection to the "islanders." No colds developed as a result of this contact by the evening of Sept. 23.

On Sept. 23, the third author arrived with five others. All six had been inoculated intranasally at Aberdeen on Sept. 19 with one of their "pedigree" virus strains from the Salisbury unit. On arrival at the island, four of these subjects had already got colds. In the other two, colds were doubtful, but they developed well later. Details regarding the cold strains and regarding the experiments to study the routes of transfer of infection are adequately given. Much to their surprise, no colds had developed in any of the 12 exposed "islanders" by Sept. 29.

After failure to obtain "takes" from exposure to induced colds, they conducted an experiment to compare the infectivity of natural and induced colds and adequate details are given of this experiment. A natural "wild" cold did spread to three out of eight subjects.

These results suggest that there is a difference between natural and artificial colds as regards infectivity. Full exposure of eight previously isolated persons to 10 induced colds, both in their incubation and fully developed stages, failed to reveal one instance of natural "jumping" of infection; on the other hand, one natural cold, even though it had reached its fifth day, and was no longer active, induced colds in three out of eight persons after an exposure of only three and one-half hours.

Louis Goldstein,[3] in a paper on cold weather as a factor in the epidemiology of grippe and the common cold, states that it is generally believed that one or more filtrable viruses are concerned in the etiology. Exposure to viral agents, however, does not necessarily provoke the clinical syndrome of grippe or colds. A lowering of resistance seems essential before enough damage is incurred by the respiratory epithelium to permit invasion by the pathogenic organisms.

Prominent in numerous accounts of the origin of respiratory illness is the factor of exposure to severe cold. This is first borne out by the seasonal variations in the occurrence of colds and other respiratory conditions. Because of this the author decided to observe a large series of children and record his findings.

The children were exposed in February and March. Approximately eight to 12 days after any day of subzero temperature, there appeared a sharp and sustained increase in the number of cases of acute illness. A second increase in the number of cases of acute illness was noted three to five days after each initial peak. Each successive subzero day was associated with a subsequent increase in the number of sick children. Following final exposure to subzero weather, the incidence of acute illness gradually declined to endemic levels.

Goldstein believes an interpretation of the data is offered in terms of anamnestic reactions substituting for clinical infection. (The anamnestic reaction is a period of rapid production of large quantities of antibodies by a previously sensitized animal or person in response to minute antigenic stimuli.) These are held to provoke the clinical syndrome of common colds, cold-equivalent or grippe as a manifestation of biochemical disequilibrium.

Fletcher and Fletcher[4] think it surprising that little attention has been paid in Great Britain to the resistance that may be gained to the common cold and its complications by adequate quantities of ascorbic acid.

They believe that no single figure for the daily requirement of ascorbic acid is likely to suit all individuals at all times.

This is likely to vary according to circumstances, as does that of common salt. Completely consistent results between various experiments, even though well-planned and controlled, are, therefore, not to be expected. They mention various reports showing a favorable effect on resistance to infection with daily doses of 90 mg. or more. They believe that the results of adequate experiments would show that considerable benefits can arise from ascorbic acid treatment given in sufficient quantities at the right time.

An experiment on the use of an antihistaminic drug for treatment of the common cold was carried out very successfully by Fisher[5] at a Summer camp for boys. There were 260 boys between the ages and nine and 16 years at the camp, together with a staff of over 100.

At the beginning of the season the campers were instructed to report for treatment at the earliest symptoms of a cold, because the chance of cure was greatest at that time. One group was given tripelannamine (pyribenzamine) hydrochloride 50 mgm. four times daily for boys 12 years old and over, and 25 mgm. four times daily for boys under 12 years. The other group was given a white lactose tablet four times daily. Therapy was continued in each case for 48 hours, and if at that time symptoms still persisted, routine symptomatic therapy was started. A good case record was kept for each patient.

If a boy reported back a second time with a cold during the Summer, he was given treatment in the opposite group to his previous one. Patients of known allergic nature were excluded from the test. Those of unreliable or unstable personality were also excluded.

As it happened, there were 37 cases in each group and the results were almost identical in each, there being 11 cured and 17 improved. Side effects were few, as only four cases complained of drowsiness in the tripelannamine hydrochloride group, but there were also four cases of drowsiness among those given the placebo.

The author concludes that from his study tripelannamine proved no more effective than a placebo in the treatment and prevention of the common cold in its early stage. He also stated that the giving of medication for the common cold, be it placebo or tripelannamine, gave satisfaction to the majority of patients.

Tremble[6] correlates some of the known facts of ciliary movement in certain lower forms of animal life with those facts known about ciliary movement in humans. The marked variations in normal human nasal mucosa and submucosa in different areas and the distribution of cilia are described.

In general it may be stated that the thickness of nasal mucous membrane is dependent upon the degree of impact and air flow over the surface. For instance, the exposed inactive anterior third of the septum and lateral wall has a thick membrane of 10 to 15 cells in depth, while the membrane in the active posterior two-thirds has a layer of only eight to 10 cells. In protected areas not influenced by direct air currents, such as the middle and inferior meatuses, the cells are four or five layers deep, and the membrane is thinnest of all in the sinuses, where the exchange of air is negligible. The mucosal lining of the sinuses consists of only two or three layers of short cuboidal cells and is often only a fraction of a millimeter in thickness. Except for the ethmoid cells, the stroma is loosely attached to the sinuses.

In other words, the more exposure to outside air laden with dust, smoke and fumes of all kinds, the thicker the epithelium; the less exposure to these influences, the thinner the membrane. The depth of the stroma containing the blood vessels is in proportion to the depth of the epithelial layer. The stroma is widest in the exposed anterior position of the nose and disappears almost entirely in the sinuses.

SINUSITIS.

Tremble[7] has written a clear, concise resumé of the conservative treatment of sinusitis, a considerable portion of which deals with sinusitis in children. He observes first that the signs and symptoms of acute and subacute sinusitis are so

indefinite at times that the infection is often not recognized in the early stages. This is particularly true in children where a postnasal discharge is frequently neglected. In infancy the sinuses are simply outgrowths of the nasal mucous membrane; therefore, infections such as acute rhinitis which affect primarily the nasal mucosa also involve the sinuses. Transillumination is a useful aid in diagnosis, but in children its scope is limited as the thin bones of the antra often transmit the light too brightly, even when filled with pus. A dull light which just shows through the bones usually gives more information than intense light; X-ray is of much greater value. In the treatment he favors vasoconstrictors used to reach the middle meatus in the lateral head low position. When washing of the antrum is not tolerated he advocates inserting a catheter into the antrum and provides illustrations of the instrument used and of the drain in position. Many other aspects of sinusitis are given in this article which is a very useful general discription of present day treatment of acute and subacute sinusitis.

Walter C. Price[8] discusses in two articles the recognition and treatment of sinobronchitis in children. He defines sinobronchitis as "involvement of the sinus membranes by an infectious process of sufficient intensity to have implanted itself likewise upon the bronchial mucosa, either directly (by means of contiguous extension or postnasal secretion) or indirectly (by lymphatic spread)." The study concerned 384 patients admitted to hospital over a three-year period. Roentgen examination was made of the chest and sinuses and 126 children showed evidence of sinobronchial disease. Three classifications of degree of respiratory tract involvement were established: the first class, or slight involvement, showed minimal thickening of the sinus membrane associated with prominent hilar nodes or faint increase in pulmonary markings; the second class, designated as moderate, showed definite membrane swelling and sinus opacity, together with accentuation of bronchovesicular markings; the third class, called advanced, showed opacity of the sinuses with sometimes polypoid changes, along with bronchopneumonia infiltration or lobar consolidation.

The prevalence of sinobronchitis in the 126 children was presented in a table which showed 105 cases in the first class, 11 cases in the second class, and 10 cases in the third class. Study by age groups showed the prevalence of sinobronchial infection was greatest in children under nine years of age, and moderate to advanced sinobronchitis was present more often in children five years of age or under.

The writer of the article says acute sinobronchitis arises from bacterial infection of the respiratory tract epithelium, and there is a rapid spread of the inflammation to the mucosa of the sinuses and bronchial passages. He describes the pathologic changes in the mucosa under three stages, according to the extent of the involvement.

In the second article, Price,[9] along with Veronica Binns, lays down a treatment program for chronic childhood sinobronchitis. Three groups were formed and the patients were divided among these in the numbers of 16, 18 and 20, respectively, there being 54 cases in all.

Treatment for Group 1 consisted of an antihistaminic drug (syrup of histadil) administered orally, and parenteral penicillin. Group 2 treatment was the same as Group 1 with the addition of intranasal aerosol penicillin inhalations. For Group 3 the treatment consisted of the antihistaminic drug, parenteral penicillin, intranasal aerosol penicillin inhalations, and parendine penicillin nose drops (par-pen). The results obtained in Group 3 were uniformly better than the other two classes after treatment was carried out for about a week in most cases. Evaluation of the results was based upon changes in the Roentgenograms made on completion of therapy in comparison with those taken at the beginning of treatment. The rationale of the combination of the above drugs is the elimination of the infectious process while simultaneously controlling the allergic response of the sinobronchial mucosa to bacterial protein. Thus the hyperplasia of the membrane regresses, permitting normal epithelium to regenerate.

Edward E. Brown,[10] in discussing rheumatic fever, says six pertinent observations must be explained and correlated. The

observations are: *1.* high incidence of preceding upper respiratory infections; *2.* increased antistreptolysin titers prior to, and at the onset of the disease; *3.* failure of tonsillectomy to prevent recurrences of rheumatic fever; *4.* variety of organisms cultured in rheumatic fever; *5.* geographical variations, and *6.* seasonal variation in incidence. He then proceeds to show that the etiologic agent is the hemolytic streptococcus, and that the focus is chronic sinusitis.

Brown says that for the effective prophylactic and active treatment of rheumatic fever, infected sinuses should be drained. This is accomplished as follows: *1.* Elevation of the head and avoidance of prolonged recumbency. *2.* Use of proper nose drops or a benzedrex inhaler twice daily, followed by hot wet towels to the face. Ephedrine sulphate 1 per cent, in normal saline solution, tends to open the ostea of the sinuses, while moist heat to the face helps to drain purulent secretions under tension. *3.* Avoidance of chilling. *4.* Allergens should be sought and avoided, since they tend to produce congested sinuses. *5.* Fatigue should be avoided.

The author claims that in man, chronic sinusitis is the only consistent focus for streptococcus toxins under pressure. Rheumatic pains result when they are forced from the sinuses into the general circulation. In his rheumatic fever patients he always notes the condition of the nasal sinuses and prescribes treatment for all who show activity. Fever, pain, and other symptoms of rheumatic fever subsided rapidly. Treatment of chronic sinusitis was continued indefinitely and was found to be an effective means of prophylaxis against rheumatic recurrences.

ANTIBIOTICS.

Davison[11] concludes that sensitivity cultures on blood agar are a useful means of determining which antibiotics should be used and the size of the dose necessary to promptly cure an infection. He has noted that the gram positive cocci causing otolaryngologic infections have become relatively resistant to the action of penicillin. Between 3,000,000 and 6,000,000 units may be required daily. In severe infections he advises

the use of streptomycin as well. He feels that topical applications of antibiotics are of little value. Aureomycin and terramycin are effective as long as vomiting and diarrhea do not interfere with their absorption.

Everett[12] reports two cases of anaphylactic reactions from the instillation of penicillin into the maxillary sinuses — these are probably the first due to local application. Both patients over a period of years had received penicillin therapy without ill effects and were probably sensitized by repeated exposure.

Wyngaarden and Seevers[13] have tabulated the common side effects of antihistaminic agents. Most of these are innocuous and are important merely because they are annoying; however, in some patients the drugs are avoided because of the unpleasant severity of the side effects.

Severe toxicity is rare, but serious reactions have resulted from idiosyncrasy or overdose. Eleven fatal cases were reviewed, in eight of which the patients were under the age of two. Infants and children seem predisposed to the development of convulsions from overdosage and the mortality rate in infants when convulsions develop is high — adults tend to development of central nervous system depression which may on occasion be lethal.

Agranulocytosis is another serious development which has been reported nine times, with one death.

ALLERGY.

Dill and Bolstad[14] report observations on 25 patients suffering from allergic rhinitis whom they treated by spraying the nose with a saline suspension of cortone acetate. They were critical in their choice of cases: two were children, 17 were men, and six women. All had had nasal symptoms for many years; several had asthma; 16 had nasal polypi; others had had polypectomies previously; 17 had been found sensitive on allergic studies. Each patient underwent a four-hour period of observation in the clinic, during which time the nose was sprayed every half hour and detailed examination followed each application. No general reactions were noted. All

patients experienced relief of nasal stuffiness and postnasal drainage. They were re-examined after a 24-hour interval and all reported they were free of any irritation and had had no local or general symptoms. The first few patients so treated were then given a quantity of the solution with instructions to spray each side of the nose four times a day at home and return for further clinical observation.

Six case reports are given. The child, age nine, two months after treatment was symptom-free. In one adult numerous polypi in both nostrils disappeared during treatment but recurred when the spray was discontinued.

It is the authors' impression that the use of cortisone nasal spray produces a temporary relief of the nasal symptoms of allergic rhinitis and they are continuing their experiment in the hope of gathering further data on the effects of the drug when used locally in the nose.

Stromme[15] feels that a number of maxillary sinus infections are primarily allergic in origin with added secondary infection. He feels that this aspect of the pathogenesis will be missed unless a very careful history is taken and all available diagnostic aids are used.

He describes a method to determine eosinophilia in antral secretion and used this cytohistologic method on 78 outpatients with pus in the antral washings. The method showed that 18 per cent had marked eosinophilia, and yet on first routine examination the presence of allergy was not suspected. Further careful history and re-examination clarified the picture of allergic maxillary sinusitis with superimposed infection.

TONSILS.

Gale[16] states that there are few subjects on which opinions are more divided than on that of tonsillectomy. He notes great variations in the prevalence of the operation in different parts of the country and proceeds to review certain statistical evidence, because such evidence is objective and is relevant to the consideration of the general indications and contraindi-

cations of operation. It must always be remembered that all the statistical evidence in the world, however carefully compiled, will not solve the problem in the individual case. Whether a child should have his tonsils out, and when, is a clinical and not a statistical problem. Part of one of the reviews ended with the words: "One cannot avoid the conclusion that there is a tendency for the operation to be performed as a routine prophylactic ritual for no particular reason, and with no particular result."

He finds the following recommendations generally accepted:

1. Tonsils should very rarely be removed before the age of five. Adenoids should be removed at any age if they are causing nasal or Eustachian obstruction.
2. The operation should not be performed on a child who has never had symptoms, whatever may be the appearance of the tonsils.
3. Tonsils should be removed on account of frequent attacks of tonsillitis with fever.
4. Tonsils should be removed at a suitable interval after a quinsy.
5. Tonsils should be removed in the very rare instances when they are so large that they cause mechanical obstruction.
6. Tonsils should be removed in chronic diphtheria carriers when other treatment has failed.

The decision whether tonsillectomy is necessary or not is by no means an easy one and both the general practitioner and the laryngologist have a contribution to make to it. The general practitioner knows how often the child has had tonsillitis and how ill he has been with it, and the laryngologist is in the best position to evaluate symptoms of nasal and Eustachian obstruction and to consider how far the child's symptoms may be due to a chronic sinusitis. Generally speaking, it is better to delay decision than to hurry it and, above all, to avoid operation on tonsils which have been recently inflamed.

Gairdner[17] comments upon that large group of potential subjects for tonsillectomy, young school children of five to seven years who suffer from recurrent upper respiratory infections. If to these are applied conservative measures, one quickly learns what the natural history of the syndrome is — that it is usually self-limited, with a marked tendency to spontaneous cure at about seven to eight years; therefore, he believes that decision about operation on this group of young school children should be postponed as long as possible. If this were done he believes many of these children would end by keeping their tonsils.

Kershaw[18] states that observation that the syndrome of recurrent upper respiratory infections in school children is self-limiting is not new. He believes it is possible to go even further and to say that investigation among younger children in nursery schools, nursery classes and day nurseries show that the phenomenon is independent of age and occurs at that point in a child's life at which he is first exposed to herd infection. The process is, perhaps, rather physiological than pathological, and constitutes a stage on the road to immunological maturity.

It seems not unreasonable to suggest that enlargement of the tonsils, like enlargement of lymphoid tissue elsewhere in the body, is a process secondary to infection in the appropriate drainage area. Mere bigness of the tonsil ought to suggest that some primary cause should be sought and treated.

Whittier[19] urges reassessment by the ear, nose and throat specialist before the operation is performed on children originally selected by him for tonsillectomy. He frequently sees children who were scheduled for operation and who, two years later, though still awaiting this operation, have had little trouble from their tonsils in the meantime, and appear to have healthy tonsils at the time of his examination. He feels that such a reassessment would result in many children having their names crossed off these long waiting lists.

He believes it is not universally appreciated that the tonsil is not a static structure, but one that can increase or decrease

in size without necessarily requiring interference. He is in agreement with the argument that simple enlargement of the tonsils is not a prime indication for tonsillectomy.

A complaint often heard from parents of children in nursery schools is the frequency of colds, and tonsils have often been removed to prevent these colds. It is difficult to see how the removal of one of the defensive weapons of the body could raise resistance to a virus infection.

Hyde,[20] from a study of 1,000 patients plus a review of the literature, points out that there is a high percentage of tonsillectomized patients who are found to have tonsil remnants—68 per cent when removal is done by general practitioners and 64 per cent when the operation has been done by nose and throat specialists.

The author points out that there are certain inherent factors which make tonsillectomy a difficult operation. The exposure is limited by the size of the mouth — the fact that illumination can only come from one direction — the difficulty of hemostasis and the fact that the usual position of the patient allows blood to obscure the line of dissection.

In his own practice the author has been able to reduce the incidence of "remnants" to 18 per cent — of which, only two of 48 were actually tonsil tissue, the remainder being lymphoid tissue without demonstrable crypts (giving a success percentage of 99 per cent).

He advocates the Davis type mouth gag for removal under general anesthesia and advises against the use of the snare.

For removal under local anesthesia he positions the patient so that he sits with his head looking downwards and forwards so that the blood runs out of the mouth rather than down the patient's throat.

Reeves and Brill[21] suggest that no adenoidectomy be considered complete without the examination for removal of diseased or hypertrophied salpingonasal, salpingopalatine and salpingopharyngeal folds. They state that the literature is re-

plete with articles advising irradiation after adenoidectomy for destruction of the remaining or re-formed lymphoid tissue. In their opinion many a radium treatment will not be necessary if we remove the hypertrophied folds during adenoidectomy. It is not at all rare that a simple adenoidectomy does not relieve mouth breathing. They found in 10 cases the enlargement of these folds was so big that removal was advisable. The elimination of big masses of lymphoid accumulations in a place so openly exposed to bacteria will diminish the number of focal infections. They describe a radical adenoidectomy under visual control.

Wright and Pray[22] present a study of 1,648 tonsilloadenoidectomies. The first 411 cases were used to study the effect of vitamin C upon secondary bleeding. In the last 1,237 cases a study was made of the effect of elimination of acetyl salicylic acid on secondary bleeding. Their conclusions are as follows: Administration of vitamin C has no appreciable effect on secondary bleeding following tonsilloadenoidectomy. Acetyl salicylic acid given after tonsillectomy increases the incidence of late secondary hemorrhage. This is shown by the marked reduction in the incidence of secondary hemorrhage when acetyl salicylic acid is withheld. This increased bleeding incidence from the use of acetyl salicylic acid may be due to its local effect in the mouth. Vitamin K taken before and after the operation appears to have no antihemorrhagic effect.

Belal[23] reports the accidental discovery, during routine microscopical examination of tonsils and adenoids, of bilharzial ova. The patient was a male, 12 years old, whose adenoids were removed because of nasal obstruction and snoring at night. The type could not be determined. No cercarie or worms were observed. Three photographs of sections accompany the report.

Slater and Stephen[24] consider endotracheal intubation the ideal method of administering anesthesia for tonsillectomy and adenoidectomy. For, today the anesthetist must be prepared to maintain an even, adequate plane of narcosis for from 30 minutes to more than an hour. They consider it best to bring the child into hospital the afternoon before surgery

and it is preferable for patients to be watched closely by the nursing and resident staff for 24 hours after operation. Thus the child spends two nights in hospital. At their hospital, the Children's Memorial Hospital in Montreal, it is the custom to give these children nembutal, morphine, and scopolamine one hour before going to the operating room. (A table showing the dosage is provided.) With this combination, patients are quiet and asleep when seen by the anesthetist and yet they will open their eyes and respond to questions. A patient receiving these drugs requires careful watching before operation to insure that his airway does not become obstructed. After operation the effect of the agent may persist for several hours and delay return of the active reflexes which aid so much in promoting a smooth convalescence. The authors consider carefully certain complications of anesthesia, such as anoxia, hypoxia, reflex laryngospasm following extubation, and anoxemia. They state that during the last four years, in which more than 3,000 tonsillectomies and adenoidectomies were done, there were no pulmonary abscesses and only one patient who developed bronchopneumonia.

[*Comment:* The authors of this resumé do not agree that the above technique is the ideal one for this operation in children. At the Hospital for Sick Children, Toronto, the number of these operations has been several times greater per annum and there have been no postoperative pulmonary abscesses for over 30 years. The reason for this excellent record is considered to be prohibition of preliminary medication, short anesthesia, and attention to the child on the operating table after operation until the cough reflex has returned and the patient has vomited. The operations are booked at half-hourly intervals.]

Barkash[25] mentions ways in which the physician can be helpful with the emotional aspects of his patient's health. He feels that all that is needed is common sense, decency, sensitivity and intuition in handling of patients.

Among other medical practices he criticizes the usual lack of preparation and explanation with regard to children undergoing tonsillectomy.

He feels that considerable emotional trauma can be avoided if the physician explains to the child what is wrong and what the child is to expect when he enters hospital for the operation. He feels the mother should be allowed to accompany the child right up to the moment of anesthesia and be there immediately upon awakening.

Anderson and Faust[26] state that because cardiac standstill continues to be an ever present danger during general anesthesia, otolaryngologists should be able to institute some predetermined plan of treatment as soon as the diagnosis is made.

Resuscitation efforts can preserve life and, as well, an undisturbed psyche in about one-third of the cases.

Adequate therapy maintains circulation and oxygenation of blood until normal activity of heart and respiratory system is resumed. Artificial circulation must be established in three and one-half minutes and cardiac massage is the most effective measure.

A case of cardiac standstill after tonsillectomy under general anesthesia is presented.

POLIOMYELITIS AND TONSILLECTOMY.

Miller[27] presents an unusually careful study of 1,229 cases of poliomyelitis which occurred in Los Angeles County during the epidemic year of 1949 in an attempt to determine whether there is any relationship between a recent tonsillectomy and adenoidectomy operation and *1.* the incidence of poliomyelitis and *2.* the development of bulbar poliomyelitis, as well as in an attempt to determine whether *3.* tonsillectomies and adenoidectomies should be discontinued during the Summer months of highest poliomyelitis incidence.

The results of the statistical analysis indicate no clinical or statistical significant deviation between the actual and the expected incidence of poliomyelitis developing in Los Angeles County during 1949 in patients recently tonsillectomized and adenoidectomized even in the months of July through October.

In view of these observations he believes that no reason was found to discontinue doing indicated tonsillectomies and adenoidectomies during the Summer months.

This study impresses these reviewers as being admirably and thoroughly carried out. This impression was shared by four discussers of his presentation. The discussions occupy three pages of fine print and are a valuable addition to the paper.

Siegel, Greenberg and Magee, from the Bureau of Preventable Diseases, New York City Department of Health, made an extensive report on the relationship of tonsillectomy to poliomyelitis. This work is under two headings: first, "Studies on Incidence in 1949,"[28] and second, "Frequency of Bulbar Paralysis, 1944-1949."[29]

In 1949, New York City experienced its third largest outbreak of poliomyelitis, there being 2,446 cases and 179 deaths. During the year, in a population of approximately 8,000,000, 53,007 tonsillectomies were performed during 1949 in New York City Hospitals, but very few of these were carried out during July, August and September. Although 74 cases of poliomyelitis occurred in recently tonsillectomized patients, only 50 cases were considered since these were operated upon in the New York City Hospitals where the total population at risk was known. Even with this reduced number of cases, they still were able to conclude that in New York City in 1949 it appeared that tonsillectomy predisposed to the development of clinical symptoms of poliomyelitis.

For the second part of their report, Siegel, Greenberg and Magee took their 1949 statistics, but also went back to 1944 and included all the years between as well. Like 1949 in New York City, 1944 was also an epidemic year for poliomyelitis. It was in 1944 that a query on recent tonsillectomy was carried out for the first time on the Health Department's poliomyelitis cards.

In this survey there were 6,524 cases of poliomyelitis, of which 1,026 were the bulbar type. Among the reported cases, a history of tonsillectomy within one year of the date

of onset of the disease was given in 194. This latter number formed the basis of the study. These 194 cases were compared with a "matched group" as a control. They found that there is a predisposition of recently tonsillectomized cases of poliomyelitis to bulbar paralysis. The relationship seems firmly established for cases occurring within one month after operation and is most marked in the first three weeks or within the usual incubation period of poliomyelitis.

MOUTH AND THROAT INFECTIONS.

Landsman, Grist, Black, McFarlane and Blair[30] present a carefully tabulated study of a properly controlled trial of 100 patients in ordinary urban practice who complained of "sore throat" as the leading symptom. The patients received tablets which, though differing in composition were similar in appearance. One set contained 0.5 gm. of sulfanilamide, another, 0.5 gm. of sulfatriad, and a third, 0.5 gm. of lactose. They have decided that the results of their therapeutic trial do not support the thesis that sulfonamides exact a beneficial effect in the treatment of the "acute throat." Those who place more faith in the virtue of clinical impressions will no doubt be unconvinced by an array of "statistics." It is well to remember that the average duration of fever and pain in those cases treated with lactose was exceedingly short — namely, 1.07 and 1.95 days, respectively. In view of this, added to the fact that in about half of the cases a streptococcal etiology is unlikely, it seems that to use drugs which have definite and known dangers is unnecessary, besides being expensive.

Macdonald and Watson[31] endeavored to find out whether sulfonamides were efficient and economically reasonable drugs to use in the treatment of acute throats in particular under Service conditions.

Eighty-two consecutive cases of acute tonsillitis were divided into two equal parts in a random manner. They were all treated by rest in bed and given copious fluids to drink. One-half were given 50 0.5 mg. tablets of sulfatriad. The other half received an equal number of lactose tablets identical in appearance to the former.

Analysis of clinical records revealed no difference between controls and treated cases in the speed of recovery from various symptoms. The medical officer in clinical charge was, however, able to make an estimate of "goodness of cure" which had a significant relationship to administration of sulfatriad.

The authors felt that the routine administration of sulfonamide to patients with acute tonsillitis cannot be supported by the results of this experiment.

The results of these two trials, says the *British Medical Journal*,[32] agree in failing to provide evidence that sulfonamides accelerate recovery from an acute sore throat. On the other hand, they do not decide whether this treatment is effective in preventing such complications of a streptococcal tonsillitis as otitis media or subsequent rheumatic fever; only one case of the former occurred in all the 182 patients considered in these two papers and rheumatic fever is mentioned in neither of them. In a sense it is more important to protect a patient against these consequences than to accelerate recovery from an illness which is normally of very brief duration.

In regard to cases of known streptococcal etiology, it is pertinent to inquire why drugs which are usually fully effective against the same organism elsewhere in the body should apparently fail to subdue it in the tonsil or the pharyngeal mucosa. Is it that recovery from these infections is usually so rapid that a true underlying therapeutic effect cannot materially accelerate it? In tonsillitis, is it that the streptococci are multiplying chiefly in crypts where the drug cannot reach them, or owing to the nature of the medium cannot act upon them? Whatever the explanation, we must accept the fact that the immediate effect of sulfonamides on streptococcal throat infections has sometimes been overrated.

Macleod[33] observes that general practitioners learn with surprise if not vexation that sulfonamides are valueless in acute tonsillar infections. Before the sulfonamide era acute tonsillitis was a severe illness with an acute phase lasting about a week. Untoward and common sequelae were acute

otitis media and acute mastoiditis. More remote and rare effects were rheumatic fever, arthritis, pleurisy and even malignant endocarditis. Since the advent of the sulfonamide era the clinical picture has changed. One to three days initiates the period of convalescence, and untoward effects are extremely rare. During the same period scarlet fever has become a benign disease. In the presulfonamide era he encountered on the average a peritonsillar abscess once weekly, while otitis media was not infrequent and acute mastoiditis not very rare. During the sulfonamide era he encountered peritonsillar abscess once and acute mastoiditis once. Until more observation is done and more data available he cannot believe that sulfonamide therapy is superfluous in acute tonsillitis.

The clinical picture of vesicular pharyngitis is described by David, Leavitt and Howitt[34] as follows: "The child, usually under five years of age, became ill suddenly with fever, malaise and anorexia. Examination revealed vesicular or aphthous lesions on the soft palate and anterior faucial pillars. The fever was of brief duration, usually less than 48 hours." Thirteen patients on whom studies were made during the Summer months of 1949 and 1950 showed a striking uniformity in their clinical picture. A virus organism was isolated from the throat secretions of 12 of the 13 patients. This organism was the coxsackie virus.

The coxsackie virus has been isolated from patients ill in Summer months from such infections as paralytic and nonparalytic poliomyelitis, Summer grippe, encephalitis, epidemic pleurodynia and fever of unknown origin.

Karelitz, King and Rubinstein,[35] working at the Willard Parker Hospital, proceeded to determine the clinical effect of aureomycin in cases of acute faucial diphtheria, and in diphtheria carriers. Good results had been obtained with penicillin and antitoxin, but the C. diphtheriae were becoming resistant to penicillin. For this reason it was decided to try out aureomycin in a similar manner.

Thirteen patients with faucial diphtheria, and 11 carriers, were treated with 25 to 50 mgm. of aureomycin per kilo per

day given in four equal doses. In addition, every case of acute diphtheria was given antitoxin as well, in appropriate doses. The acute cases averaged five days for negative cultures as compared to an average of 21 days in those cases receiving antitoxin alone, as shown by previous hospital records.

The carrier cases did not produce such good results on aureomycin. Of these, two developed negative cultures in one day, one in two days, one in three days, and one in 10 days. Four of the other cases were infected with nonvirulent strains of C. diphtheriae. These patients were discharged from hospital. The other two cases were treated extensively and one cleared on dihydrostreptomycin. C. diphtheriae were not eliminated from the other carrier.

The results here were satisfactory, and indicate the possibility of using aureomycin along with diphtheria antitoxin in cases of acute diphtheria, and alone in cases of diphtheria carriers.

IRRADIATION.

The clinical value of radium or Roentgen therapy of lymphoid hyperplasia of the nasopharynx has been established over a period of years in thousands of cases. The paper by Garland, Hill, Mottram and Sisson[36] discusses the relative advantages and disadvantages of X-ray and radium therapy in the treatment of lymphoid hyperplasia of the nasopharynx that causes deafness and other allied auditory disturbances.

They show that with Roentgen therapy the dosage throughout the whole nasopharynx is more homogeneous and efficient than with intranasal radium applicators. They also show that the dose in the immediate proximity to radium applicators is 10 times the dose ordinarily regarded as reasonably safe for the treatment of nonmalignant conditions. The maximum tissue dose required for cure when X-ray therapy is used is lower and consequently safer.

[*Comment:* The claims made in this article are in such disagreement with much previous work that they should not be accepted without very careful study.]

Roentgen therapy in lymphadenosis and sinusitis in children is discussed by Harold Levy.[37] He defines lymphadenosis as a condition characterized by chronically infected hyperplastic lymphoid tissue of the Eustachian tube, nasopharynx, pharynx, adenoid, tonsil, and cervical, retropharyngeal and perihilar glands. He presents a 10-year follow-up of 349 cases.

Children differ from adults in the abundance of lymphoid tissue in the nose and throat regions, which may swell during an acute infection and cause obstruction. It is for this reason that upper respiratory infections in children are so often complicated by otitis media, acute sinusitis, cervical adenitis and bronchitis. These regions may be treated successfully by radiation therapy, either alone or in conjunction with other procedures. The indications for radiation therapy are:

1. Lymphadenosis.
   a. In the young child (under five years of age).
   b. In children of all age groups with Eustachian tube obstruction (as evidenced by diminution of hearing), or granular pharyngitis, chronic or recurrent cervical adenitis, etc.
2. Sinusitis (subacute or chronic) where the usual therapeutic aids (penicillin, etc.) have failed, but before surgery is contemplated.
3. Lymphadenosis with or without sinusitis.
   a. In the chronically ill child (cardiac, tubercular, etc.).
   b. In the "nonallergic" asthmatic child.

In treatment, the author has used relatively small doses of radiation and has carried on weekly treatments for five to 10 weeks, depending upon the severity of involvement and the clinical improvement. Some patients show much greater shrinkage of lymphoid tissue than others. He considers radiation therapy preferable to radium because it is easier to administer, there is no need for anesthetic, local or general, there is a more widespread effect and the dose may be more accurately calculated.

No untoward effects were noted in the series of cases under discussion, but every safeguard must be utilized.

The results obtained in the 349 cases showed 70 per cent good, 18 per cent fair, and 12 per cent poor.

SURGERY.

Owens[38] contributes a simply and clearly described and well illustrated operative technique for the correction of choanal atresia by the transpalatine approach, which is recommended and should be studied in the original.

Although congenital occlusion of the posterior nares is an uncommon cause of asphyxial attacks in the newborn infant, it constitutes a real emergency when it does occur. The choanal atresia may be unilateral or bilateral, and may be caused by bone or membrane. Nasal breathing is difficult in the unilateral case and impossible in the bilateral one.

According to Medovy and Beckman,[39] the diagnosis of choanal atresia is made by watching the infant breathe, by the passage of a catheter through the nose, and by the instillation of lipiodol into the nose followed by Roentgen examination. A newborn infant will try to breathe through the nose and this may give rise to blue-spells, particularly at feeding time if occlusion of the posterior nares is present.

A one-sided obstruction case may be left alone until the infant is older, but bilateral occlusion usually requires early attention. An opening is made through the obstructing bone or membrane, and then the opening is dilated once or twice a week for awhile. This allows the infant to carry on well and to be fed properly.

These cases of choanal atresia, as a cause of asphyxia in the newborn, are not common, but should be kept in mind. The pathologist also should think of this cause when an autopsy is performed on a case of sudden asphyxia death.

Beyer, Blair and Lipscomb[40] state that the treatment of intranasal meningoceles has been far from successful. Only three cases reported in the literature have ended in recovery.

They report an intranasal meningocele in an infant 22 months of age. Two photographs of radiograms illustrate the report. A left transfrontal craniotomy was performed. Near the anterior tip of the left frontal lobe a small stalk of arachnoid-like tissue was encountered, sutured and cut. A dehiscence in the cribriform plate was easily palpated. A free pericranial graft was placed to cover the sectioned end of the arachnoidal stalk and the bony dehiscence. Fifty-one days after operation the patient was considered well.

Lewin and Cairns[41] have written a usefully illustrated article on fractures of the sphenoidal sinus with cerebrospinal rhinorrhea. It reviews their experience with 84 adults with cerebrospinal rhinorrhea following closed head injury. This condition occurs also in children and consequently this most informative paper deserves the study of the readers of this resumé.

In a series of 84 cases of C.S.F. rhinorrhea following closed head injury, a dural tear over the sphenoidal sinus was found in 11 (13 per cent) and was the only tear in relation to the paranasal sinuses in five (6 per cent).

The main clinical characteristics of these five cases was the profuseness of the rhinorrhea, due to the fact that the tear involved the overlying cisterna chiasmatica. Another sign which may be helpful in diagnosis is the radiographic demonstration of air in the cisterna chiasmatica as in one of these cases.

Neighborhood signs, such as evidence of damage to the hypothalamic region, optic chiasma, or carotid artery, were present in nearly half the sphenoid cases, but by themselves they do not necessarily indicate a fracture involving the sphenoid sinus. The preservation of loss of sense of smell is likewise no clinical guide.

Operative repair is urgently indicated. The risk of spread of infection intracranially into the basal cisterns is appreciably higher than with fractures involving the other paranasal sinuses.

Operative repair is difficult and requires a bifrontal flap, usually with division of the falx and sacrifice of one or both olfactory tracts. Three patients underwent successful dural repair and have since been followed for nearly four years. There has been no further rhinorrhea or intracranial infection and all are well or working.

Beatty[42] states that in cases where a cleft palate has been closed with good result the physician is often reluctant to have a tonsillectomy done, fearing a resultant speech defect—but when such a patient is suffering local or systemic complications from infection in tonsils or adenoids a tonsillectomy and adenoidectomy must be done regardless of the previous palate operation.

He considers that if tonsillectomy is done in the usual way an unpleasant speech defect and difficulty in swallowing will develop in most of these cases because he thinks the natural contraction of the tonsillar fossa will cause shortening by contraction of the soft palate.

The author advises delay until it is a proved necessity—preferably until the child is six or seven years old.

He describes a plastic procedure after removal of the tonsils in which the uvula is partially removed and the palate actually extended by suturing the two palatoglossal muscles together for a short distance downwards.

[*Comment:* This is an interesting proposal but the results would have to be seen before it could be approved.]

TUMORS.

Kerwin,[43] in reporting a case of juvenile nasopharyngeal angiofibroma in a white youth of 16 years of age, presents a concise review of the whole subject. This is valuable because it has been the privilege of very few men to see several of these interesting cases. These tumors occur during the pubescent stage, usually at about 14. The word juvenile used in the descriptive title of this condition is incorrect; rather it should be called adolescent nasopharyngeal hemangiofibroma.

It is a relatively rare tumor restricted apparently to adolescent males. It regresses as maturity (20 to 25 years) is reached. Epistaxis is the chief symptom and usually is serious. Ligation of the carotid is of questionable benefit. Secondary extirpation is almost impossible. Radiation and hormone (androgen) therapy offer the best possibilities of control. Large tumors producing deformity of skull (frog-face) are resected by electrocoagulation and remnants or recurrences are treated with interstitial irradiation and androgen. The most conservative therapy is always employed because of the usual spontaneous regression of the tumor at maturity.

Finermann[44] presents a study of juvenile nasopharyngeal fibroma in a 13-year-old girl. This tumor is a benign, firm neoplasm found primarily in the nasopharynx of young adults. Onset of the neoplastic growth always occurs during adolescence. Spontaneous regression is said to occur frequently at sexual maturity or adulthood. The girl whose case is presented had not reached the age at which such neoplasm may tend to regress spontaneously. She had a prominent smooth, pink encapsulated mass behind the left tonsillar fossa, extending into the hypopharynx. Elevation of the palate revealed the mass to be merely the apex of a pear-shaped tumor filling the entire nasopharynx. The tip of the mass was removed for section and three capable pathologists independently were of the opinion that the structure was identical with that called juvenile nasopharyngeal fibroma. Six weeks after X-ray therapy was initiated, the neoplasm had receded high into the epipharynx, the projection in the left side of the nose had receded and shrunken. Symptomatically, too, much improvement was evident. Six months after the child was first seen her improvement has continued. The author intends to delay surgical excision as long as possible.

Penha[45] reports from Portugal the successful treatment of a nasopharyngeal fibroma in a 12-year-old boy with the nettle Urtica membranacea. This is a common plant used by the natives to stop bleeding. The patient had his nasal fossa filled with a fibrous tumor having a smooth surface and bleeding easily; also, the right choana was filled with a peduncu-

lated tumor and on the right cheek there was a hard tumor the size of a pigeon's egg. As the patient's anemia was progressing without any response to the therapeutic measures, a blood transfusion was given. Removal of the mass was unsuccessful owing to serious hemorrhage. The tumor contained extensive areas of fibroblastic cells and abundant intercellular cement. X-ray treatment was without benefit. The anemia became progressively worse in spite of much therapy. It was while forced to give transfusion daily that the author decided to give the diluted infusion and later the aqueous extract of the Urtica membranacea. All the symptoms, hemorrhage, nasopharyngeal tumor and nodule of fibrous tissue in the cheek subsided. The patient was allowed to go home approximately 11 months after his condition was first noticed. Recently the patient has stopped the treatment without showing any after-effects or recurrences.

The author describes the characteristics of the plant and the preparation of the solutions he used. He implies that his report is only the beginning of an interesting study.

Hirst and Charland[46] discuss lymphosarcoma of the tonsil and report on two cases, one of which was a child four months old. Tonsillar lymphosarcoma is characterized by marked hypertrophy of one tonsil, which may be two or three times normal size. The tumor presents a dull hyperemic appearance with rounded contours and a nodular surface. It is soft and cystlike, and often becomes ulcerated and bleeds easily. In the differential diagnosis, infectious adenopathy and lymphatic diathesis must be considered; in these conditions there is enlargement of lymph nodes of moderate degree, and they are not so progressive in their extension. In these conditions pain is much more pronounced.

The patient was hospitalized on April 27 because of high temperature and a draining left ear and after treatment with sulfadiazine, myringotomy and X-ray therapy was discharged on May 5. During and subsequent to the aural infection, the anterior cervical lymph nodes were enlarged and there was slight enlargement of tonsils and adenoids. On June 14, the

patient had no ear or nose symptoms or increase in adenopathy or tonsillar enlargement. On July 19, there was noted for the first time the mass in the right tonsil and an increase in the size of the right cervical nodes. Adenotonsillectomy was postponed because the history suggested pertussis. On Aug. 9, the mass was much larger and not so hyperemic and the veins of the pharyngeal mucosa could be seen extending over the surface of the mass. A punch biopsy specimen was taken. It was the opinion of the pathologist that the probabilities pointed to lymphosarcoma. The patient was then treated with intraoral and external X-ray therapy and on Aug. 23, after having received half of the total irradiation, the mass was definitely smaller and not so hyperemic. On Oct. 6, the patient was readmitted to hospital for study, treatment and a tonsillectomy if condition permitted. The chief complaints were sore throat, mouth breathing worse at night, weakness and extreme pallor. The tonsillar mass seen Aug. 14 had now apparently diappeared. On Oct. 6, blood cell count showed 2,580,000 red cells, with 48 per cent hemoglobin and 37,700 white cells. Another blood cell count on Nov. 13 showed 1,850,000 red cells with 26 per cent hemoglobin and 100,000 white cells. In spite of blood transfusions the child died Nov. 19 after a few peaks of temperature to 105° with a final diagnosis of acute lympholeucemia.

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GRASSES AS FOREIGN BODIES IN THE BRONCHUS AND LUNG.

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Grass is the name applied to any plant of the family of Poaceae. The family is a large one, and it includes all of the relatively few common cereals. It is characterized by flowers borne in spikelets of bracts, called glumes.

Incidence. — Considering the ubiquity of grasses, and especially the fondness of children for playing with them, their occurrence as foreign bodies in the tracheobronchial tree is extremely rare. Both medical literature and clinical experience support the foregoing statement as to rarity. At the clinic, up to the present writing, there have been only seven cases of grasses as foreign bodies in the air and food passages.[12] Of the seven cases of grasses, all were in the tracheobronchial tree. As to medical literature, there are a number of valid reasons why every case occurring is not represented in print; but making a reasonable allowance for such unpublished cases, there can be no question as to rarity. Seydell[25] comments on this rariety in relation to the perforative type of grass head, and the clinical records above given show that the rarity applies to the lodging type in the tracheobronchial tree. Seydell found only nine cases of the perforative type in the literature. To these he added one case encountered by himself, making a total of 10 cases of spontaneous extrusion of grasses. Adding to these 10 cases the 18 additional cases of extrusion found in the literature and herewith presented in the following table (see Table 1) makes a total of 28 of the extrusive type of cases. With the just mentioned seven cases of the lodged type, the total is only 35 cases of inspirated grasses as foreign bodies in the tracheobronchial tree.

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TABLE 1.

CASES OF INSPIRATED GRASS SPIKES EXTRUDED THROUGH THE THORACIC WALL.

Reported by	Foreign Body	Sex—Age	Duration*	Result
Ayral[1]	Wild barley	M—13 yrs.	25 days	Recovery
Deloriere and Balident[3]	Foxtail grass	F—5 yrs.	15 days	Recovery
Foster[4]	Millet grass	? ?	Approximately 12 days	Recovery
Johnson[16]	"Barley grain"	F—2 yrs.	About a week	Recovery
Knapp[17]	"Grain spike"	? 8 mos.	About 3 weeks	Recovery
Martin[19]	Spike (Barley?)	F—14 yrs.	6 weeks	Recovery
Purcell[20]	Head of grain	F—4 yrs.	29 days	Recovery
Resch[21]	Ear of grain (Kornähre)	F—9 mos.	10 days	Recovery
Riccioli[22]	"An oat" (Spikelet)	M—15 yrs.	? ?	Recovery
Schubert[23]	Cereal spike	M—9 yrs.	9 days	Recovery
Sehring and Shaw[24]	Spike, Foxtail grass	M—4 yrs.	14 days	Recovery
Carrière[17a]	Grass spike	M	18 days	Recovery
Destrangeş[17a]	Grass spike	3 or 4 yrs.	6 weeks	Recovery
Labath[17a]	Grass spike	F—16 yrs.	7 days	Recovery
Le Boeuff[17a]	Grass spike			Recovery
Pincent[17a]	Rye spike	1 yr.	24 days	Recovery
Vigla[17a]	Grass spike	F—1 yr.	3 or 4 wks.	Recovery

*Duration refers to the elapsed time between the inspiration and the extrusion of the foreign body, as nearly as could be determined from the report.

Etiology and Prophylaxis. — As with most other cases of exogenous foreign bodies in deeper air passages, accidental inspiration of parts of grasses is due to putting inedible substances in the mouth. In adults this is carelessness on the part of the patient. In case of older children it is usually carelessness on the part of parents in not teaching children the dangers of putting small inedible substances in the mouth. In case of babies and very young children carelessness in allowing parts of grasses and especially spikes of grass to be

within reach. They should not be allowed to play or crawl on the floor until such objects, or any other small objects, have been swept away or otherwise removed. If a baby is to be placed on the lawn, a large sheet should be spread for him to play on and even then he should be watched. Games and other play with grass spikes should be forbidden.

PATHOLOGY.

Clinical evidence indicates that there are two types of pathologic progress in cases of grass spikes occurring as foreign bodies in the tracheobronchial tree. Seydell suggested that grass heads have the "power of spontaneous self propulsion." In the discussion that followed, Clerf[2] stated that he had not seen a case of spontaneous perforation by a grass head, but that in a number of cases of timothy grass head there seemed to be no tendency to perforation. All were removed bronchoscopically. This is also the experience of the clinic. Though the total number of cases of parts of grasses reported as foreign bodies in the deeper air passages is small (35 found in literature) pathologic progress and the clinical features justify the postulate that there are two distinct classes of cases. One class may be appropriately called the *lodging type* and the other the *one-way travel type.* Clarity of presentation seems to call for a summary of the well known clinical facts relative to one-way travel of pointed foreign body toward the periphery of the lung, and for a discussion of the new problem of the botanical features of different grass spikes of the same genera as those that have been reported as having been extruded through the thoracic wall.

LODGING TYPE OF GRASS SPIKE AS A BRONCHIAL FOREIGN BODY.

The timothy spike is typical of the lodging type. No case of spontaneous perforation by timothy spike has been found in the available literature. The seven cases of this lodging type reported[12] presented definite characteristics which may be summarized as follows:

1. In all cases the foreign body was a spike (panicle) or part of a spike of timothy grass *(Phleum pratense).*

2. All of the patients were children; the respective ages were eight, 11, 20 months; three, six, nine, 14 years.

3. Right bronchial tree, five cases; left, two cases.

4. Duration of sojourn of timothy spike in bronchial tree before bronchial removal, eight, eight, 21, 25 days; three and one-half, nine months.

5. Pathologic conditions were as follows: vegetal bronchitis; tight bronchial obstruction to ventilation and drainage; suppuration; intermittent bringing up of pus accumulated in drowned-lung area.

6. Pus of foul odor in long-sojourn cases.

7. The symptoms were: irregular fever; weight loss or failure to gain weight and to grow; cough; bringing copious amounts of usually foul pus, at intervals.

8. Diagnosis was by bronchoscopy and removal of the foreign body. Bronchoscopy was clearly indicated by the drowned lung. In the case of the 20 months old baby there was a clear statement of the parents that the first coughing attack was when the then 11 months old baby was crawling on the floor. This always implies a tentative diagnosis of foreign body; but in this case it was ignored for nine months, while the baby was treated for pneumonia, tuberculosis and thymic tracheal compression.

9. The timothy spike presented itself to bronchoscopic view in six cases; a small fragment of a spike was hidden out of sight in one case. In no case did a cylindric part of a spike of timothy make enough travel toward the periphery to disappear from the bronchoscopically visible lumen.

10. Treatment. — In all the cases the timothy spike was removed bronchoscopically. All patients recovered perfect health generally as well as locally in the bronchial tree.

11. The *prognosis* of the lodging type of grass spike apparently is good. There was no mortality or serious complication in any of the seven cases. The recovery, general and local in the lung, was complete. This was remarkable in some of the cases; for example, the 20 months old baby, who for nine

months had a timothy spike *(Phleum pratense)*, 3.3 cm. long by 0.5 mm. thick, in the tracheobronchial tree, with copious bringing up of foul pus from the area of the drowned lung. The recovery after bronchoscopic removal was prompt.

ONE-WAY TRAVEL TYPE OF GRASS SPIKE AS A BRONCHIAL FOREIGN BODY.

Pathologic Postulates. — Autopsic data are lacking. The only death reported was from bilateral pulmonary tuberculosis, which was evidently in an advanced stage prior to the inspiration of "an ear of wild oats." Though the cases of perforative grass spikes are rare, sufficient clinical evidence has now been accumulated to justify certain postulates. Clinical case reports demonstrate the fact that spikes of certain grasses when inspirated into the tracheobronchial tree can be forced on through the bronchial tree, pleural layers, intercostal tissue, dermal layers and be thus extruded. In most of the cases there was no empyema and no pneumothorax; these two clinical facts justify the postulate that a preparatory adhesive pleuritis had sealed the visceral and parietal layers of the pleura together at the site of subsequent perforation. These and other features indicate that these cases differ from cases of pulmonary abscess discharging into the pleural cavity and followed by chronic intercostal fistula. In typical cases, so far as can be determined from the reports there was no large pulmonary abscess formation, nor prolonged suppuration. The passage was rapid, ranging from eight to 29 days in children; no chronic suppurative track remained in the lung. Though bronchoscopies are not reported to have been done subsequent to the extrusion of the grass spike, the reports of prompt healing and recovery of perfect health imply that there were no suppurative or bronchiectatic sequelae. There seems to have been prompt healing of the entire track of transit in the wake of the one-way travel. This is supported by the evidence in the one case in which bronchoscopy was done during the one-way travel of a spike of grass (see under Bronchoscopic Appearances). In two of the exceptional cases it seems that there was residual suppuration due to operative

opening into the pleural cavity by an ill-advised enlargement of the extrusive orifice to improve drainage of a supposed pulmonary abscess, which was evidently only an area of drowned lung, such as cleared promptly in other cases after extrusion.

Mechanism of One-Way Travel of Foreign Bodies in the Bronchi. — The ratchet-like, one-way travel of foreign bodies in the bronchi was first noted in relation to pins (Jackson, 1915[5]). A number of illustrative cases were reported (Jackson and Jackson[8]), in which the pin had been worked downward by the ratchet-like action of the respiratory and tussive elongation and diametric luminal enlargement, followed by shortening and diametric diminution of the bronchial lumen. In some of the cases the head of the pin "outdented" the visceral pleura. Again, in writing of staples,[11] the same authors state, "Their branches, in most instances, diverge at a small angle, directing the points toward the walls of the invaded passage. . . . The shape of the foreign body (small rounded head and divergent pointed extremities) favors working deeper and deeper into the tracheobronchial tree by a ratchet-like action. Under the enlargement and diminution of lumen, and elongation and shortening of the bronchi at each respiratory cycle and at each cough, the head slips down easily, but upward movement of the foreign body is prevented by the points sticking into the bronchial wall; the result is that the foreign body is forced down into the smallest bronchus it can enter."

In this one-way travel, as in all other phases of bronchial obstruction, the laws of physics apply. As stated by Jackson,[14] "Movement relates the bronchi to that part of science known as mechanics; which treats of the action of forces on bodies, solid, liquid and gaseous. So long as the bronchi contain only gaseous bodies they are concerned with the laws of pneumatics. When they contain liquid bodies they are concerned with the laws of hydrostatics and hydrodynamics. The presence of solids brings into consideration the fundamentals of mechanics in its restricted sense." One of these fundamental laws is that when sufficient force is applied to a movable solid body, the body moves in the direction of least resistance. For

example, a schooner close hauled does not blow off to leeward because of great lateral resistance of the hull. It moves in the direction of least resistance, which is within about four points (45 degrees) of the direction of the wind, thus reaching a point to windward. Conforming to the same law of physics, a foreign body whose points, by engaging in the bronchial wall, stop retrograde movements, moves in the direction of least resistance. This, in the lung, is peripheral.

The foregoing now generally accepted clinical facts explain the mechanism of one-way travel. It remains to determine the nature of the greater penetrating forces manifested by certain grasses. In considering the penetrative one-way travel of certain grass spikes, it is a primary essential to study the botanical structure of the grass spikes of the same genera as those of grass spikes reported to have made the one-way transit and extrusion.

One difficulty encountered in the study of the botanical structure of the particular penetrative grass spikes is that, unfortunately, most of the case reports of grasses do not identify the foreign body. When the botanical classification is recorded, study is simple. For example, Larget, Maurice and Lamare identify the foreign body in their case report as a head of grass, *Trisetum flavescens*. This is the type species of the genus *Trisetum*. The name is from the Latin *tri*, three, and *setum*, bristle, alluding to the awn and the two teeth of the lemma. These three bristles are the important factors to be considered in the mechanism of one-way travel as discussed in subsequent paragraphs.

The reports of more than half the cases give no details sufficient to classify the foreign body. For example, "a head of grass," "head of common grass," "head of wild grass." As there are many genera and hundreds of species of grasses, indigenous and imported, in various countries of world, classification from such names is impossible; moreover, common names are often applied to a number of different genera, even in the same locality. Only one case report gives the botanical name of the foreign body, *Trisetum flavescens*, as just mentioned. Of the others, some of the common names

given can be classified with some degree of accuracy as to genera if not as to species, from names given in the case reports. These are as follows: "ear of barley, 3 cases; "spike of wild barley"; "head of rye"; "wild oats"; "head of Foxtail grass," two cases; "millet grass"; "an oat" (spikelet?); timothy. "Cereal spike" and "head of grain" may have been any of the cereal grasses herewith illustrated or other less common genera.

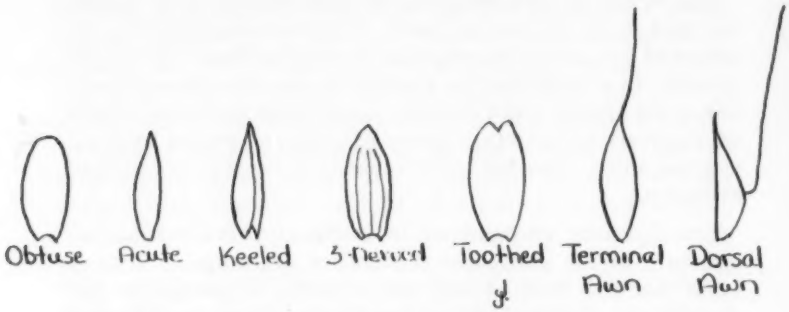

Fig. 1. Sketches of the botanical features of spikes and spikelets of grasses germane to the subject of grasses as foreign bodies in the bronchial tree. The glumes are attached at the base; they extend upward and diverge slightly outward. The upper free end may be obtuse or acute, toothed or awned. The lower of the two glumes enclosing the flower is called the lemma. An awn is a bristle-like projection from a glume and collectively is commonly called a beard. Glumes and awns point upward in growth; when inspirated into the tracheobronchial tree the points trail. The glumes, lemmas and awns are the pawls in the ratchet-like mechanism of one-way travel and perforation of the thoracic wall by certain grass spikes.

For clarity of presentation of the subject of the mechanism of one-way travel, penetration, perforation and extrusion of grass spikes, it seems best to select from the foregoing list the genera that best illustrate the subject. For this purpose an illustration of a fair average normal spike of the respective genera obviously is better than a photograph of the specimens removed, because the latter are incomplete and are the worse for wear in being forced through tissue; moreover, only a few of the specimens are illustrated in the reports. The illustrations that have been published, however, have been carefully checked in comparison with the botanical sketches herewith reproduced (see Figs. 1 to 6).

The natural function of the grass spike is to produce flowers, expose them to shaking in the wind for pollenization, produce seed and protect it to maturity. The seed loosens and drops out. The remaining parts of the spike hold together in their natural places for a time, retaining the form of a spike. They are sketched separately in Fig. 1. No mention of seed is made in the case reports. In one case there is mention of a "green head of barley." It may have contained immature seed.

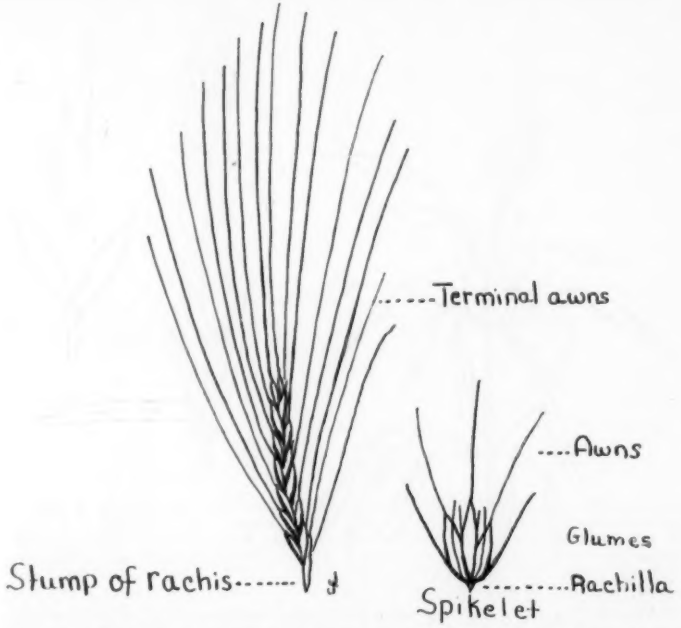

Fig. 2. Sketch of an average spike and spikelet of cultivated barley (Hordeum vulgare). Length, 2 to 10 cm. exclusive of awns; glumes divergent, tapering into a stout awn; awn of lemma straight, 10 to 15 cm. long. Barley spike reported as perforating in four cases. No case of lodgment appears among the cases found in the literature.

From the viewpoint of the physicist the subject of the one-way travel, perforation and extrusion of certain grass spikes

naturally falls under three heads, namely: *1.* the stops against retrograde travel; *2.* the source of power; *3.* mechanisms applying the power to the work.

The Ratchet-like Stops Against Retrograde Travel of Certain Grass Spikes. — It seems certain that a detached grass spike, or part of one, or a spikelet, when it enters the tracheobronchial tree, must enter and travel with the stub of the rachis (or rachilla in case of a spikelet) leading and the glumes and awns trailing (see Figs. 1 and 2). This postulate is supported by *a.* the mass of clinical evidence of the fact

Fig. 3. Sketch of panicle and of spikelet (enlarged) of cultivated oats, *Avena sativa*. Spikelets are large and suspended in a spike-like panicle; glumes about 2 cm. long, lemma bidentate; awns straight and stout, rarely wanting. Report of perforation by "an oat" (spikelet) in one case.

that practically all pointed foreign bodies, such as bar-pins, open safety-pins and staples, enter the tree and travel with the point or points trailing; *b.* the appearance of the few illustrations of specimens of perforative extruded grass spikes

that have accompanied case reports; *c.* a study of the structure of average grass spikes of the same genera and species as those reported as having perforated the chest wall (see Figs. 2 to 6) will convince anyone that the stops against retrograde travel of such grass spikes in a bronchial tube, or in tissue, would be the upward divergent glumes and awns and that these would trail; *d.* palpation of any one of these grass spikes gives through the sense of touch unequivocal

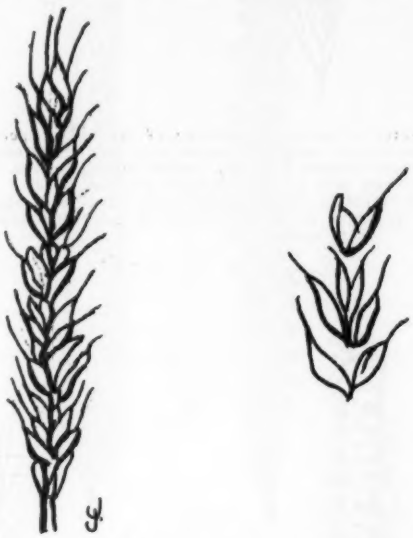

Fig. 4. Sketch of spike and spikelet (enlarged) of wheat (*Triticum aestivum* or allied species). Spike 5 to 12 cm. long. Spikelets placed flatwise; glumes rigid, keeled, apex abruptly mucronate or toothed or with one to several awns; lemmas broad, keeled, asymetric, abruptly pointed or awned. In the "beardless"' wheat the awns are short and in some, spikelets wanting, but the spikes are not entirely awnless. Probably some of the four perfoating foreign bodies reported as "head of grain," "cereal grain," "cereal spike," "grain spike" were a spikelet or part of a spike of wheat.

evidence of the stopping efficiency of the glumes and awns; *e.* obviously, therefore, if any adequate moving force is applied to such a foreign body, it must conform to the laws of physics and move in the direction of least resistance, like a close-hauled schooner, as before mentioned.

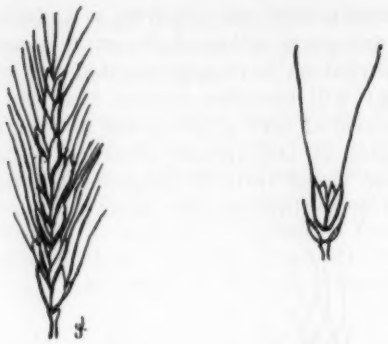

Fig. 5. Sketch of spike and spikelet of rye (Type species, *Secale cereale*). Spikelets are placed flatwise against the rachis; glumes, narrow, rigid, pointed; lemmas broader, sharply keeled, ciliate on the keel and exposed margins, tapering into a long, resilient awn. A head of rye has been reported as a perforating foreign body in one case, and probably was also the foreign body in one or more of the cases of "head of grain," "cereal grain," "cereal spike," "grain spike."

Fig. 6. Sketch of spike and floret of the type species of foxtail grasses, *Alopecurus pratensis*, "meadow foxtail." The name of the genus is from the Greek alopex, fox, and oura, tail, alluding to the cylindric bristly panicle. There are 10 species classified botanically, but they vary little from the type species in the structural features that relate to one-way travel and perforation. Panicle 3 to 7 cm. long, 7 to 10 mm. thick; glumes 5 mm. long, villous on the keel and pubescent on the sides; awns exserted 2 to 5 mm. Foxtail grass is reported as the foreign body in two cases of perforation and extrusion through the chest wall.

In the bronchi the direction of least resistance is, obviously, peripheral. When bronchiolar and parenchymal tissues are reached, a point of least resistance is created by suppuration

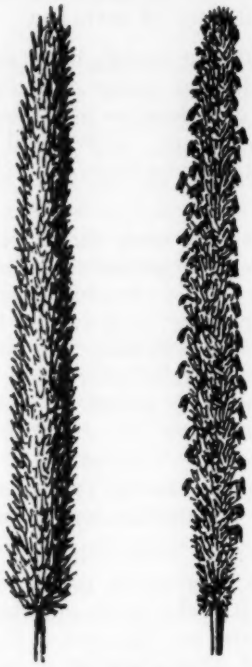

Fig. 7. Sketch of an average spike-like panicle of timothy grass *(Phleum pratense)*. Panicle cylindric, 5 to 10 cm. long, sometimes longer; the spikelet small, crowded, spreading; glumes equal, membranaceous, one, sometimes 3 mm. long, truncate, awn 1 mm. long, sometimes absent; pectinate or ciliate on the keel. Empty glumes awn pointed. These glumes, lemmas and short awns are relatively soft and lack resiliency when wet. At the right is shown a timothy panicle in full bloom. The rough appearance is due to the stamens which dangle in the wind for pollenization. They are soft and could not be concerned in stopping retrograde travel, nor much in mechanical irritation. Allergic reaction a possibility. Seven cases of timothy spike are reported as foreign body lodged in the bronchial tree. None perforated. All were removed bronchoscopically.[12]

in advance of the rhythmic prodding of the rachis. This is evident from the clinical fact that suppuration precedes extrusion of the grass spike through the skin. It is only a

small suppurative area that promptly heals after its task of creating a point of least resistance is completed. It should be emphasized that this suppurative track is not pulmonary abscess. The emphasis is important clinically to avoid ill-advised surgery at the site of extrusion.

Bronchoscopists who do foreign body work will recall frequent experiences with two phases of endoscopic images that are germane to the consideration of one-way travel of grass spikes and spikelets, namely: *1.* bronchial movements; *2.* behavior of foreign bodies under force of respiratory and tussive movements.

The bronchoscopist has seen the fence staple slip away deeper and deeper in the bronchial tree, because of the visible ratchet-like stopping by the upward projecting points of the staple serving as a pawl by sticking into the bronchial wall. He will, therefore, easily visualize the powerful and direct resistance to retrograde travel afforded by the many upward and divergently projecting points of glumes and awns of perforating grass spikes.

THE SOURCE OF POWER FORCING TRAVEL OF GRASS SPIKES OR SPIKELETS.

There is a tradition in some European communities that grass spikes have a mysterious power of movement. One source of this belief is probably the entertaining trick of laying a spikelet of *Avena sterilis* (one of the species of "wild oats") on a plate and, after a prelude of patter "the touch of the magic wand" is used to "bring the spikelet to life." The contortions simulate the struggles of a living insect. The secret is that this species of the genus *Avena* has twisted awns and when the spikelet is moved by the wand onto a wet place previously arranged on the plate, the absorption of moisture causes the awns to jerk and move weirdly in untwisting. Taking this trick seriously, it may be said that it could not be considered as evidence of a source of power for perforative extrusion of a foreign body for four reasons: *a.* the movement of untwisting ceases in a few minutes, whereas the shortest recorded period required for travel and extrusion is

seven days; *b.* the untwisting of the awns is not a propulsive movement; c. no case of extrusive perforation due to *Avena sterilis* has been published; *d.* none of the genera identified in the published case reports of perforative grass spikes have twisted awns.

Another tradition that may have helped to create the belief that grass spikes have a mysterious power of movement is the common saying among English country boys to the effect that "barley grass heads will crawl." The knowing boys put the spike upside down under the wristband of the shirt, and it progresses upward and arrives at the shoulder.[26] Deloriere and Balident[3] mention the same kind of play as customary among the children of Picardie, using the spike of foxtail grass.

Palpation of any of the grass spikes of the same genera as those reported to have been extruded through the chest wall reveals a remarkable degree of compressibility and resiliency of the spike as a whole. It has been suggested that this springiness could be the power that causes the grass spikes to perforate the chest wall. The springiness is due to the spikelets, especially the glumes and awns; however, neither the awns nor any other part of a grass spike generates any power.

From the point of view of the physicist a spring generates no power; it simply delivers the power that has been stored in it from some source of power.

The *source of all the power* that drives the grass spikes through the bronchi, pulmonary parenchyma, adherent pleural layers, intercostal and subcutaneous tissues is muscular. It comes not merely from the bronchial muscles, but also from that powerful group of muscles whose action is synchronized by the autonomic nervous system in the operation of the respiratory bellows. Working at the average rate of, say, 20 cycles per minute throughout the 24 hours, with additional and even more powerful tussive cycles, it is no wonder that a spike can be driven through in as short a period as eight days, in some cases.

MECHANISMS APPLYING THE POWER TO THE WORK.

In the application of this power to the work of driving the spike or spikelet through the bronchopulmonary and intercostal tissues, five mechanisms are concerned.

1. Tussive Squeeze. — An important part of the mechanism in the application of the power to the work of driving the perforating grass spike through the lung, on the way to extrusion is the tussive squeeze.[9] The powerful muscular compression in the creation of the bechic blast far exceeds that of the partially passive diminution of the expiratory phase of the respiratory cycle. Physiologically, this powerful squeeze forces secretions and exudates up from the periphery to the larger bronchi where the bechic blast can blow it out. If a grass spike, in the right lower lobe, for example, were not stopped by the spikelets, glumes and awns, the powerful squeeze would force it up also; but the spike being stopped, the lobar tissues are forced up against the stub of the rachis, which is thus caused to penetrate the tissues. This action can be better understood by an experience of my childhood. I was playing with a soft, old-fashioned pin cushion that had no pins sticking in it. In carrying it, I squeezed it with my hand and the point of a needle emerged through the fabric and stuck in my hand. The needle had evidently, at some previous time, been pushed into the cushion until the blunt, eye end was under the containing fabric. Then it travelled one way in course of handling of the pin cushion, as needles do in living tissues by force of muscular movements. This well known one-way needle travel in tissue by force of muscular movements is illustrative of the perforation of a grass spike through the chest wall. The "case" of the pin cushion illustrates the forcing of the grass spike through the smaller bronchi and pulmonary parenchyma by the mechanism of the tussive squeeze.

2. The Bechic Blast.—The blast of air itself exerts no penetrative power on the grass spike; but the greatest power in driving the grass spike through the lung and thoracic wall is exerted by the powerful contractive pressure of the chest wall, as it is forcibly compressed in creating the bechic blast.

The mechanical principle involved is similar to the "case" of the pincushion mentioned. It is also similar to that of the seamstress pushing the fabric over the needle which is stopped against retrogression by the thimble.

3. The Stub of the Rachis as a Perforating Tool.—Some of the reports of cases have photographic halftone illustrations of the foreign body specimens. These illustrations all show a sharp end of the stub of the rachis (or rachilla in case of a spikelet). Most of the grass spikes found detached under natural conditions on farms have a sharp stub; not often sharply pointed, but sharp on palpation. Many stubs have multiple points of fracture that could introduce infection. The relatively small diameter of the end of the stub of most grass spikes would permit entrance into a bronchus 1 mm. in diameter with little force. It may be postulated that the stub of the rachis is the perforating tool. It seems possible that if a staple had a pointed instead of a rounded head it might travel deeper than it does.

4. The Spring Mechanism of the Spikelets Glumes and the Awns. — The resiliency of the spikelets glumes and awns noted on palpation has been mentioned. Though this resiliency generates no power, it may be postulated that power is stored in these springy parts of the perforative grass spikes, from the compression of the expiratory diametric diminution of lumen, and to a much greater degree during the powerful compression of the tussive squeeze.

The bronchoscopist will have a mental conception of the appearance of, *a.* a forcing of the grass spikes in the direction of least resistance by compression; *b.* a compression of the resilient awns, and *c.* followed by a springing deeper of the whole foreign body as the compressed awns are released simultaneously with the inspiratory elongation and diametric enlargement of lumen ahead.

5. Post-Tussive Inspiration.—Another mechanism by which the thoracic muscular power is applied to forcing the one-way travel of the grass spike in the tracheobronchial tree is synchronous with the tussive squeeze. This mechanism is the

deep inspiration that always follows cough. It is preparatory to another cough, in case of a paroxysm. The bronchoscopist, doing bronchoscopy without suppression of the cough reflex, is quite familiar with the remarkable luminal dilation of this *postbechic* or *intertussive deep inspiration.* The area of cross-section is more than doubled. Most foreign bodies advance toward the periphery during this extraordinary luminal dilation, accompanied as it is by the inrush of inspiratory air. Gravity helps but little, if at all, in case of a grass spike.

6. The Prodding of the Stub of the Rachis.—Another important mechanism in application of thoracic power to the forcing of one-way travel of grass spikes is the prodding of the stub of the rachis (or the rachilla in case of a spikelet). The bronchoscopist from his abundant accumulation of moving endobronchial images is familiar with the rhythmic expiratory compression of the right lower lobe, for example. This compression is, of course, accompanied by firming and diminution in size of the lobe and by shortening of the invaded lower lobe bronchus. With a grass spike firmly fixed against retrograde travel in this bronchus, the perforating sharp leading point of the spike (stub of the rachis) and the tissue come together. According to the physical laws of mechanics they could not do otherwise. This movement is rhythmic prodding of the tissues by a perforating point, and is necessarily followed by a progressive advance in the position of the grass spike. This prodding and the progressive advance of the grass spike are much more forcibly exerted and greatly accelerated at intervals by the tussive squeeze.[9]

For clarity the penetration of the lung is used for illustration. When the pleura is approached the inflammatory reaction in advance of the prodding of the rachis seals together the two layers of the pleura. These and the aponeurosis, intercostal muscle and subcutaneous tissues are perforated by the prodding of the rachis, carried on by the movement of the thoracic muscles, just as muscular movement forces a needle to travel through tissue. The only pathologic difference is that the needle is subject to ionization which keeps it relatively sterile; whereas the stub of the rachis carries on a lim-

ited area of infective suppuration which creates a point of least resistance in advance of its arrival. This is probably because the stub of the rachis is not as good a perforating tool as a pointed needle.

In the *calculation of the efficiency* of the just mentioned mechanisms of application of the thoracic muscular power to the work of driving the perforative grass spike, full count must be taken of the *frequency of the application of the power.* For illustration, respiratory cycles may be rated at 20 per minute. Calculating 1,440 minutes in the 24 hours, we have 28,000 daily applications of the power relative to respiratory cycles. The powerful tussive squeeze is an additional and an enormously greater application of power, but it is variable and difficult to estimate as to its frequency. It certainly is frequently applied during the one-way travel of a grass spike.

Failure of Timothy Spikes to Perforate. — Although spikes and spikelets of grasses are peculiarly the kind of foreign body recorded as perforating from a bronchus to the dermal surface of the thorax, it is noteworthy that timothy spikes (panicles) have not been recorded as perforating, though they are recorded in the literature in seven cases, which is a greater number than any other genus of grass mentioned by name.[12]

Speaking of timothy heads, Clerf[2] stated, "These were all removed from the bronchi. Timothy heads have small barbs and their propulsive movement seems to be lost as soon as the smaller bronchi are entered." Our experience at the clinic coincides with that of Clerf.

As before mentioned, a search of the literature reveals no case of spontaneous perforation by a timothy spike-like panicle. In one of the clinic cases a small part of a timothy spike had reached a segmental branch of 2 mm. luminal diameter, but it was found bronchoscopically and removed. It was the small size of the fragment that explained its reaching a segmental branch.[12]

Botanical examination of a timothy panicle under magnification, when dry and also when thoroughly moistened, yields

ample mechanical reasons fully to explain the clinical fact that they have not been recorded as having been driven through the lung in the way that certain other grasses have been driven. They are worked peripherally as far as the smallest bronchus that will accommodate them without trauma; but no farther. The average timothy spike-like panicle and its spikelets do not have the structure for the efficient pawl-like action for conversion of reciprocative movements into one-way travel.

The spikelets of timothy are crowded tightly together into a spike-like panicle (see Fig. 7). The glumes, flowering and empty, are translucent, membranaceous, small, short, thin, weak; they are acute and end in an awn-like point about 1 mm. long, not bristly, and sometimes absent (see Fig. 7).

Palpatory stroking of an average timothy spike when dry yields a sensation of slightly more roughness when stroked downward than when stroked upward; but the roughness is nothing like the resistance felt on downward stroking of an average sample of any of the perforative grass spikes. The latter are relatively smooth on upward palpation ("upward" and "downward" refer to the living position of the grass spike). When compressed between finger and thumb and then released, the awns and glumes of all the perforative grasses transmit a remarkable degree of efficiency in delivery of stored force of compression. The resiliency of the perforative spikes is little, if at all, diminished by wetting. By contrast with the perforative grasses, the palpation of an average dry specimen of timothy spike reveals little resiliency and when wet it is relatively soft and limp.

Bronchoscopic Appearances. — In the cases of lodged timothy panicles seen within a few days after inspiration there was an acute vegetal bronchitis. In cases of long sojourn, the proximal end of the timothy panicle could be seen submerged in pus, of which the odor was foul. After aspiration of this purulent accumulation, slight oozing of blood from granulations was noted. After aspirating again, the proximal end (top) of the spike could be seen buried in granulation tissue. Search of the literature has revealed no record of the appear-

ance of a perforative grass spike *in situ*. The only reported case in which bronchoscopic examination was made while a perforative grass spike was in the lung is that of Foster and Cody.[4] The grass spike (millet grass) had disappeared. The pathologic conditions noted were mucosal inflammation and a source of hemorrhage. The spike was extruded through the chest wall a few days later.

Symptoms of Grasses in the Bronchi and Lungs. — These symptoms are those common to all foreign bodies in these regions. The initial symptoms are choking, gagging, coughing and dyspnea; they are practically never absent, but may be unobserved, ignored, unelicited or overlooked.[7,10,12,13,15] In almost all the published cases of grass spikes they were unelicited. In more than half the cases they were volunteered by the relatives when shown the specimen bronchoscopically removed or spontaneously extruded. Wheezing respiration (asthmatoid wheeze) usually accompanies the initial symptoms, but is not so commonly noted by lay relatives. Then a symptomless interval intervenes. After that, the symptoms are those of the various common diseases of the lungs, such as productive cough, purulent sputum, often blood streaked, and, after a time, usually foul. Soon the typical symptoms of chronic pulmonary sepsis develop, particularly chilliness, sweats, irregular fever, weight loss, or oftener failure of the child to gain in weight. Pulmonary abscess is rare, but drowned lung is common. In the baby who had a timothy panicle in his right bronchus for nine months (from 11 to 20 months of age) there was intermittent discharge of accumulated foul sputum from the obstructed and drowned lower lobe bronchial tree. As often occurs in right lower-lobe foreign-body cases, in some of the perforating grass spike cases the patient had the symptom of tense right abdominal muscles, misleading to a diagnosis of appendicitis. Subjective sense of presence is usually absent or not subjectively localizable. Two symptoms are common: *a.* pain in the chest, not definitely localizable by the patient, and *b.* hemorrhage. Both of these are rarely, if ever, absent in case of perforating grass spikes.

Doubtless the pleuritic type of pain is present for a short time and is localizable in these cases, but it is not mentioned in the case reports.

Diagnosis. — A working diagnosis of foreign body can be made in a child if a history of the just mentioned initial symptoms can be elicited by close questioning of the parents. A diagnostic bronchoscopy will then be decisive in a case of the lodging type of grass head. In the perforative type, however, bronchoscopy might be negative from the one-way-travel disappearance of the spike. In one such case (Foster and Cody[4]) the source of hemorrhage was noted. After the symptomless interval, suppurative symptoms call for a diagnostic bronchoscopy as part of a systematic complete diagnosis in any patient with pulmonary symptoms. The *physical signs* early may show hyper-resonance or an impaired percussion note, according to whether obstructive emphysema or obstructive atelectasis is present. Soon, however, drowned lung develops and dullness, almost always basal rather than apical, is present. This dullness is usually mistaken for pulmonary abscess or pneumonia; both of which are rare in foreign body cases. Differentiation from pneumonia is easily made by the absence of the typical auscultatory signs of lobar pneumonia as understood by the internist. The published case reports show that too much importance has been given to the physical signs of percussion and too little to those of auscultation.

Grass spikes are, of course, radioparent, but *Roentgen examination* is of utmost importance. It should be done in every child with a history of the just mentioned initial symptoms, and also in every child with any pulmonary symptoms. Grass spikes may show Roentgen signs of atelectasis or emphysema early; later, shadows of drowned lung appear, usually in a lower lobe, most frequently in the right. This appearance has been misinterpreted as pneumonia or abscess, but the experienced radiologist of today will interpret it as drowned lung.

Differential Diagnosis. — Nearly all the published case reports show some of the pitfalls in differential diagnosis. In addition to the just mentioned misinterpretations of pneumo-

nia and pulmonary abscess, there were reported erroneous diagnoses of diphtheria, chronic bronchitis, bronchiectasis, asthma, tuberculous rib and pulmonary tuberculosis. In two cases a misinterpretation of hard right-sided muscular tension misled to a diagnosis of appendicitis. Other pitfalls were in overlooking, ignoring of failure to elicit diagnostic symptoms of foreign body in the tracheobronchial tree.

TREATMENT.

Of seven cases of the *lodging type* of grass spike (panicle) the foreign body was bronchoscopically removed in all and all the patients recovered perfect health, general and local, without any sequelae; therefore, it may be said that no treatment other than bronchoscopic removal need be considered. Long continued drowned lung in the lodged grass spike cases may call for a few subsequent bronchoscopic aspirations to expedite clearing of the tree.

In case of a child with the initial symptoms of choking, gagging and coughing in surroundings that justify suspicion of inspiration of a grass spike, diagnostic bronchoscopy should be done at the soonest possible moment, because of the possibility of an inspirated grass spike of the one-way travel type. Minimum elapsed travel time to extrusion in seven and eight days in some of the cases implies the possibility of a spike getting beyond the bronchi quickly, because this bronchial part, obviously, is the speediest part of the journey. These postulates are supported by the previously mentioned case reported by Foster and Cody,[4] in which bronchoscopy, done two or three days after a spike of millet grass had been inspirated, indicated that the spike had already disappeared on its one-way travel.

In case of a presumably perforative type of grass spike, or spikelet, that, because of its radioparency, has disappeared from reach of bronchoscopy, spontaneous extrusion should be awaited. This postulate, however, is subject to the opinion of the physicians and surgeons concerned in the care of the par-

ticular patient. "Presumably perforative type," as indicated by published clinical records, means all grass spikes other than timothy.

So far as can be determined from the published reports of cases, no after-treatment was necessary in the typical perforative cases. In the management of the dermal extrusion abscess, conservatism is of utmost importance. All radical debridement, enlargement to improve drainage, and like surgical measures are contraindicated. When the perforative spike presents itself, it can be lifted out and a sterile gauze pad can be held in place with adhesive strips. Evidently barriers against spreading infection protect the adjacent tissues, and prompt healing follows the spike on its one-way travel and extrusion.

PROGNOSIS.

A remarkable feature of grass spikes as foreign bodies in the tracheobronchial tree is the absence of mortality and the prompt recovery of the patient from a serious pulmonary condition after the bronchoscopic removal, or the extrusion, of the foreign body.

For example, in the *lodging type* of grass spike, a 20-month-old baby carried a timothy spike-like panicle 3 cm. long in a foul area of drowned lung nine months with only intermittent natural peroral drainage, yet recovered rapidly and completely after bronchoscopic removal.[12]

In the cases of the *perforative type* of grass spike the healing of the track following extrusion, the prompt healing of the dermal abscess and the rapid systemic recovery seem fully to justify a favorable prognosis.

In 34 cases of inspirated grass spikes there was no mortality due to the foreign body. The only death was from a bilateral pulmonary tuberculosis that was already at a serious stage before inspiration of the grass spike. The transit of the grass spike did not seem to affect the progress of the tuberculous lesions. The patient did not succumb to the pulmonary tuberculosis until nine months after the extrusion of the grass spike.

SUMMARY.

1. Considering the ubiquity of grasses, and especially the fondness of children for playing with them, their occurrence as foreign bodies in the tracheobronchial tree is extremely rare. Including the 18 additional cases herewith tabulated, the total number of cases found in the literature is 35.

2. Published case records and botanical examination of normal grass spikes and spikelets of the same genera as those reported indicate there are two distinct types of cases, dependent upon the genus of the grass inspirated.

3. In one type (seven cases) the spike remained in the bronchus until removed bronchoscopically; in one case it had remained nine months in a 20-month-old baby. In the other type (28 cases) the spike was driven through lung and thoracic wall and was extruded on the dermal surface. In most of the cases this was completed without empyema or pneumothorax. These are not cases of pulmonary abscess rupturing into the pleura. They are a morbid entity. Empyema following pneumothorax was created by ill-advised surgical enlargement of the fistula in search of an abscess, in some of the cases.

4. One-way ratchet-like travel of a bronchial foreign body toward the periphery has often been noted in cases of pointed objects such as pins, bar pins, open safety-pins and staples. The greater driving force and penetrative efficiency in case of grass spikes are fully explained on the basis of the laws of physics and the botanical characteristics of the genera and species of the spikes recorded as having perforated the thoracic wall. The source of all the power that drives the spike is the thoracic muscles. The five mechanisms of application of this power are fully discussed.

5. Attention is called to pitfalls in diagnosis and postulates for guidance in clinical work are given.

6. As a contribution to medical science, it is urged that the botanical classification of the spike or spikelet be determined and included in all future case reports of grasses as foreign bodies in the tracheobronchial tree.

7. Prognosis is good. Of a total of 35 cases, the only recorded death was from pulmonary tuberculosis that was in an advanced stage when the foreign body was inspirated. Even in this case the patient (an adult) did not succumb to the pulmonary tuberculosis until nine months after extrusion of the grass spike.

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THE DIFFERENTIATION BETWEEN SYMPTOMS REFERABLE TO THE OTOLITH ORGANS AND SEMICIRCULAR CANALS IN PATIENTS WITH NONSUPPURATIVE LABYRINTHITIS.

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In describing the symptomatology of patients with labyrinthitis a distinction is usually drawn between symptoms referable to the auditory and non-auditory portions of the labyrinth. Little attempt is made, however, to extend this differential symptomatology to the two components of the non-auditory labyrinth, namely, the semicircular canals and the otolith organs. This report represents an attempt in making such a separation. It is based mainly on a comparison of symptoms observed before and after unilateral labyrinthectomy in five patients with non-suppurative labyrinthitis; the spontaneous symptoms prior to treatment are compared with symptoms induced by differential stimulation of the otolith organs and semicircular canals following recovery.

During the past few years many experiments have been carried out at the U. S. Naval School of Aviation Medicine[1] wherein healthy persons were subjected to angular acceleration, the physiological stimulus to the sensory elements of the semicircular canals, and to a change in direction of resultant force, the physiological stimulus to the otolith organs. In the

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course of these studies the differential symptomatology referable to these organs of special sense was observed and may be briefly summarized.

Among the cardinal symptoms following stimulation of the sensory elements in the horizontal canals are *1.* nystagmus, *2.* feeling of rotation *3.* and the apparent motion of objects. If more than one pair of canals are involved, the result of Coriolus' accelerations, nausea and vomiting are readily induced. These symptoms are well known, and, for the most part, have been fully exploited in the clinic.[2] The feelings of rotation are characteristic; with eyes closed one has the illusion of rotating about an axis which passes through the body and the feeling that the chair or other supporting structure likewise rotates. With eyes open the attention is chiefly centered on objects in the visual field. The impression is gained that these objects are moving but that they are not displaced very far in space. In other words they appear to drift without escaping from the visual field. In addition to the cardinal symptoms mentioned the subject always has difficulty in walking and may have symptoms referable to many other bodily systems.

Stimulation of the sensory elements in the otolith organs was accomplished by means of a human centrifuge. The subject was exposed to a change in direction of resultant force with respect to himself; angular accelerations were small and were present only during the onset or cessation of rotation. The symptomatology was assumed to be characteristic of stimulation of the otolith organs although other sensory elements were also stimulated.[1d] The cardinal symptoms include *1.* an apparent change in body position, *2.* the illusion that the supporting structures are tilting, and *3.* an apparent movement of objects in the visual field. The apparent change in position consists of a feeling of being tilted away from the vertical. This feeling of displacement contrasts sharply with the feeling of apparent rotation following stimulation of the semicircular canals. The subject also feels that the chair or other supporting structures tilt with him. Objects in the visual field appear to move and to assume a new position which

accords with the apparent change in body position. The apparent displacement is the striking feature and apparent movement is observed only with a change in the magnitude of displacement. The absence of nystagmus and nausea is noteworthy. Other symptoms are related to the magnitude of the force employed.

The symptomatology referable to the semicircular canals and otolith is very different and it is relatively easy to detect these differences in the symptomatology of the cases now to be described. It should be emphasized that this brief report is not intended to provide a full account of labyrinthine symptomatology in health or disease but is limited to those aspects which have come under our personal experience.

CASE REPORTS.

Case 1: C. E., a woman 41 years of age, was unusually well and strong until April, 1949, when she developed mumps. Convalescence was complicated by tinnitus and loss of hearing in the right ear. There were no further symptoms until six months later when she was awakened one night feeling nauseated and dizzy. Subsequently she suffered numerous attacks. They were characterized by a sudden onset of vertigo, nausea and vomiting. The vertigo consisted of a feeling of rotation to the left and objects appeared to move to the left. The velocity of apparent movement varied from "slow to fast." The most helpful treatment was to remain absolutely quiet; even a slight movement of the head aggravated the symptoms and walking was impossible. There were no illusions of tilting; this applied both to bodily position and to objects in the visual field. The attacks usually lasted about an hour. Between attacks she complained of nervousness, tinnitus and the dread of future attacks.

Many forms of therapy were tried without avail and a right labyrinthectomy in the manner described by Day[3] was carried out in July, 1950. Following operation she had severe symptoms for three days, then gradually improved but experienced difficulty in walking for six months. Recovery was complete save for tinnitus and deafness referred to the right ear.

During July and August, 1951, she volunteered to act as a subject in experiments designed to test the response to stimulation of the horizontal canal and otolith organs. A comprehensive medical examination at that time revealed no significant abnormality other than a total loss of hearing and failure to respond to caloric stimualtion in the right ear. The symptoms induced when she was subjected to a change in direction of resultant force on the human centrifuge were similar to those experienced by a healthy young person and did not resemble the spontaneous symptoms she suffered prior to operation; however. when she was subjected to angular acceleration the feelings of rotation and the illusory movement of objects in the visual field, although normal responses, closely resembled the spontaneous symptoms she experienced in the past.

Comment: The spontaneous symptomatology in this case was clear-featured. Tinnitus and deafness were referable to the auditory system. The feelings of rotation, and the apparent rotation of objects in the visual field closely resembled the symptoms induced following stimulation of the sensory receptors in the semicircular canals. There were no spontaneous symptoms resembling those induced when the otolith organs were stimulated.

Case 2: R. H., a woman 27 years of age, suffered her first attack of dizziness in the Spring of 1947; prior to that time she was well and strong, save for a radical mastectomy in 1945. At first the attacks were infrequent and between times she felt well; however, they gradually increased in frequency and their duration ranged from a few minutes to three hours. In addition to tinnitus and deafness the attacks were characterized by vertigo, nausea and vomiting. The vertigo consisted of the illusion of rotating to the right about an anteroposterior axis and of objects in the visual field rotating in the same direction. The velocity of the apparent rotation was "very fast." She sought relief by lying still. Eventually she noticed tinnitus and nausea even between attacks and was fearful of leaving the house. Various forms of therapy afforded little or no relief and a right labyrinthectomy was performed in March, 1949. Following operation she had severe symptoms which gradually decreased over a period of two months. Following convalescence she had no further attacks. Save for difficulty in orientating a source of sound she had no complaints. More specifically, she did not experience any disturbance of equilibrium when walking by day or night or when in swimming.

She was a participant in the experiments carried out during the Summer of 1951. At that time a comprehensive medical examination revealed a total loss of hearing and failure to respond to caloric stimulation in the right ear. She had normal hearing in the left ear but an abnormally decreased response to caloric stimulation, especially of the vertical canals. The symptoms induced following angular and centripetal acceleration were within the normal range. She stated that there was a close correspondence between the spontaneous vertigo and that induced following stimulation of the semicircular canals, but that the symptoms induced following stimulation of the otolith organs had no counterpart in the spontaneous symptomatology.

Comment: The findings in this case closely resemble those in Case 1. The most noticeable difference was that the apparent rotation was in the frontal rather than the horizontal plane. This could be explained by involvement of different canals.

Case 3: M. W., a woman 46 years of age, had suffered from earache on numerous occasions over a long period of time. Five years previously following a severe cold, she developed deafness and persistent tinnitus in the left ear. One year later, after eating a large amount of chocolate, she suffered her first attack of nausea and vertigo. The vertigo consisted

of both visual and postural illusions. The visual illusion was characterized by the apparent tilting and displacement of objects in the visual field. The tilting occurred gradually, never suddenly, and the slope was downward on the left. The maximum degree of vertigo was approximately 45 degrees and after a period of minutes or hours, "things gradually returned to the level." She would "stare and fight" to prevent this phenomenon but with little or no success. She denied having had the illusion that objects rotated or that they moved rapidly. Coincident with the visual illusion she felt that she was "sliding off things." This tendency was worse with eyes closed and she would clutch onto bed or chair. She discovered that this illusion of sliding was reduced if she was inclined backward about 45 degrees and remained quiet. Movements of the head might aggravate an attack. Often she was unable to walk and was forced to crawl around the house.

Little benefit was received from medical treatment, and labyrinthectomy on the left side was performed in February, 1949. Recovery was slow, but within six months she was able to resume her usual way of life. Since that time she has felt well and there has been no recurrence of symptoms.

Medical examination in August, 1951, revealed no significant abnormality other than total loss of hearing in the left ear and failure to respond to caloric tests on this side. Her response to tests on the human centrifuge were within the normal range, and she stated that the feeling of being tilted and the tilting of objects in the visual field were similar to those she experienced during spontaneous attacks of vertigo prior to operation. When subjected to angular acceleration she observed the apparent movement of objects in normal fashion but experienced no definite feelings of rotation other than a slight sensation of "sliding."

Comment: In contrast to the two cases already described the spontaneous symptomatology in this case corresponded more closely with symptoms induced following stimulation of the otolith organs rather than the semicircular canals; however, an indication that the latter were also involved is supported by the fact that the feeling of "sliding" was experienced spontaneously and following stimulation of the semicircular canals.

Case 4: P. W., a woman 45 years of age, was never seriously ill until July, 1948. At this time she first noticed ringing in the ear and members of the family noticed she was slightly deaf. The tinnitus resembled the after-tone of a bell and was pulsatile in character; she believed it was synchronous with the heart beat. A few days later, following a wedding festival, she suddenly developed headache, nausea, vomiting and vertigo. The vertigo was characterized by both visual and postural illusions. Objects in the visual field appeared to drift rapidly to the right and at times vision was blurred. In addition the entire visual field appeared to be sloping downward on the left. The degree of slope varied and at times was "pronounced." With eyes closed she had the feeling that "things were going around" but that she was motionless. Rarely she noticed a slight sensation of tilting, but this was never sharply defined. The attacks varied in duration but often lasted for hours. Walking was impossible during an attack and she was fearful to leave the house between attacks.

Various forms of therapy were tried without avail and in April, 1950, a right-sided labyrinthectomy was performed. She recovered slowly and in four weeks was able to walk. Save for two mild attacks she has had no subsequent complaints other than tinnitus and deafness. Medical examination revealed no definite abnormality other than a total loss of hearing on the right and absence of any symptom following caloric stimulation on this side.

Her responses to a series of tests involving selective stimulation of the semicircular canals and otolith organs were within the normal range. She reported that there was a resemblance between the spontaneous and induced symptoms and that this was especally true in the case of symptoms related to semicircular canal stimulation.

Comment: The spontaneous symptomatology in this case was certainly related to the semicircular canals and probably to the otolith organs as well. The former predominated and correlated better with the induced symptoms.

Case 5: Z. F., a woman 47 years of age, had suffered from dizzy spells for eight years. The only important illness prior to that time was pulmonary tuberculosis, discovered at the age of 28, which was arrested following treatment. At the age of 39, one day while eating, she felt "something pop" in the right ear. This was followed by a feeling of pressure which never entirely disappeared. After that day she noticed that on rotating the head toward the right she had a feeling of "tumbling" in that direction. This was accompanied by malaise but not by vomiting. This recurred off and on for a year, after which she was nearly free of symptoms for 18 months. Thereafter the attacks increased in frequency and severity despite many different forms of therapy. The symptomatology varied from one attack to another and even during the course of a single attack. With eyes open, objects would appear to drift to the right, sometimes slowly but often rapidly. This apparent movement was always in the same direction and might "come and go for hours." In addition there were occasions when the entire visual field seemed to slope downward to the right. The degree of slope varied greatly and at times "things were almost on end." With eyes closed, she had strong feelings of clockwise rotation about the longitudinal axis. When seated she sometimes felt as though she were tilted backward and to the right. When lying she sometimes felt as though she were being slowly tilted backward, then forward, and sometimes from side to side. These illusions were accompanied by nausea but rarely by vomiting. Between attacks, in addition to tinnitus and deafness, she usually had a feeling of "floating" and instability.

Inasmuch as all forms of medical therapy were of little value, a right labyrinthectomy was performed in July, 1950. Recovery was slow, but eventually she was greatly improved and served as an experimental subject in the Summer of 1951. Medical examination at that time revealed a well developed and nourished person. The positive findings were as follows: Teleroentgenogram of the chest revealed pathological changes consistent with an inactive tuberculosis process. Nystagmus, with fast component to the right, was readily induced when gaze was directed about 20 degrees from straight ahead. There was total deafness in the right ear, and caloric tests on that side provoked no response. The Romberg test was slightly positive on some but not all occasions. Tests designed to stimulate selectively the semicircular canals and oto-

lith organs were carried out at times when she felt well, and the responses were usually within the normal range. She readily associated the induced symptomatology in both instances with certain of the spontaneous symptoms and said they were "much the same."

Comment: Although the spontaneous symptomatology in this case was severe, extensive and variable, yet it was possible to distinguish certain symptoms which were shown by later tests to be related to both sensory organs of the non-acoustic labyrinth. It is also noteworthy that nystagmus induced in the four directions of gaze was characterized by the fast component to the right and this agreed with the visual illusion of objects drifting in this direction.

DISCUSSION.

It has long been known that the inner ear contains three organs of special sense which correspond to different physical stimuli. Since the time of Goltz[4] a sharp distinction has been made between the organ of hearing and the so-called organs of equilibrium, and as the result of investigations of Mach,[5] Breuer[6] and others, a distinction is made between the static and dynamic portions of the non-acoustic labyrinth; nevertheless, the exact rôle of the semicircular canals and the otolith organs has not yet been fully elucidated, although recent studies have contributed to our knowledge, both with regard to the physiological mechanisms[7] and the psycho-physiological effects of stimulation.[8] Much more is known concerning the effects of stimulation of the semicircular canals than of the otolith organs for the following reasons: In the case of the semicircular canals the physiological stimulus is angular acceleration, which is absent when a person is at rest but provided under limited controllable conditions; hence differences between the two states can be easily studied. In the case of the otolith organs, which respond to the force of gravity or to rectilinear acceleration, the sensory elements are stimulated even when a person is at rest, and controlled changes in stimulation are produced with difficulty. This difficulty can be overcome only if a human centrifuge is available.

Only those persons who have experienced the effects of angular and rectilinear acceleration under proper conditions in the laboratory can have a firm appreciation of the postural and visual illusions which are induced. These illusions are extremely characteristic,[1,6] leaving no room for confusion. The differential symptomatology includes much more than the perception of the illusion, and we have emphasized the fact that nystagmus is never referable to stimulation of the otolith organs and that nausea is usually referable to stimulation of the semicircular canals.

Keeping this differential symptomatology in mind, it is possible, in some cases of labyrinthitis, at least, to distinguish between certain symptoms which originate in the semicircular canals and otolith organs. Because the sensory receptors for both organs are in a single labyrinthine system it is to be expected that both might be stimulated by a common pathological factor. In the five cases described above, spontaneous symptoms referable to the otolith organs predominated in one case and were present in two others. This suggests that symptoms referable to the semicircular canals are more common, but it is necessary to add, that until the symptomatology referable to the otolith organs is more widely appreciated, cases in which these symptoms prevail may be missed.

It is only to be expected that differences will be found between the spontaneous symptoms of patients with labyrinthitis and symptoms induced in healthy persons or even in patients between attacks. This is due in part to the fact that the spontaneous symptoms result from pathological stimulation and in part to the fact that the induced symptomatology has not yet been fully explored. It would be worthwhile, in studying patients during an attack, to inquire closely regarding illusions of tilting and to determine the accuracy with which they estimate the horizontal or vertical position. The latter test should be carried out by having them adjust a luminous rod in a dark room or a rod placed before a uniform background. A fuller appreciation of symptoms which may arise from disturbances of the otolith organ should lead to a better recognition of certain cases of labyrinthitis.

SUMMARY.

Five patients with symptoms characteristic of non-suppurative labyrinthitis were partially or wholly relieved following unilateral labyrinthectomy. After recovery from the operation they were subjected to both angular and rectilinear accelerations which stimulated respectively the sensory elements in the horizontal pair of semicircular canals and otolith organs. The symptoms induced following stimulation of the semicircular canals were feelings of rotation and the apparent rotation of objects in the visual field. The chief symptoms induced by a change in direction of resultant force, which included stimulation of the otolith organs, were an apparent tilting of the body and the apparent displacement of objects so that they appeared to assume a new position in space. When their spontaneous symptoms were interpreted in the light of the induced symptoms, it became evident that the disturbances in equilibrium might be mainly referable either to one or both portions of the non-acoustic labyrinth. The spontaneous disturbances were mainly referable to the semicircular canals in two cases, the otolith organs in one, and to both sensory organs in the remaining two.

We are grateful to Prof. Hallowell Davis for his help in initiating the study, to CDR Earl Wolf, MC, USN, for carrying out caloric tests, and to Lt Vernon Bragg, USN, for audiometric examinations. It is also a pleasure to acknowledge the technical assistance of Miss Frances Clopton, Clayton S. Ezell, HMC, USN, D. I. Herrett, HMC, USN, and the staff at the Acceleration Unit at the Naval School of Aviation Medicine. Above all we are grateful to Mrs. Pauline M. Webb, Mrs. Zella O. Foutz, Mrs. C. Lucille Edwards, Mrs. Rosa Jean Haas and Mrs. Charles H. Worheide who served as experimental subjects.

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RADIOACTIVE IODINE IN THE DIAGNOSIS AND TREATMENT OF LINGUAL THYROID ADENOMA.*

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Lingual thyroid is uncommon. Its diagnosis without biopsy has not been feasible until the recent development of radioactive isotopes. By the use of radioactive iodine and the directional scintillation counter the distribution of thyroid tissue can be clearly established. It is thus possible to locate abnormally situated thyroid tissue and to eliminate thereby other possibilities in the differential diagnosis of masses at the base of the tongue. It can help to decide whether medical treatment or surgical treatment — or both — are to be carried out.

The following case report presents an enlightening history, demonstrates the use of radioactive iodine preoperatively, describes the easiest type of operation for removal of large tumors at the base of the tongue, records the postoperative radioactive studies, and suggests a modification of part of the theory regarding the development of the thyroid gland.

Case 741486: On June 20, 1951, a 30-year-old white, divorced woman entered the Massachusetts Eye and Ear Infirmary because of dysphagia. During the past six weeks the dysphagia had seemed to be increasing. On exertion there was difficulty in getting the breath, and the patient had more trouble breathing when lying down than when erect. She had noted that her voice was nasal.

These symptoms were not entirely new. She first noted difficulty in swallowing about five years ago. Periodically she had attacks that her doctor called sore throats. Often adenitis was present. On several occasions penicillin had been give to cure the "sore throat." Because of this difficulty in swallowing and breathing, an inhalation anesthetic was deemed unwise five years ago. Instead, intravenous anesthesia was used for salpingectomy and appendectomy. At least four different physicians had employed a tongue depressor to examine her throat at various times

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in the past five years. Except for inflammation, no abnormality had ever been detected. She had had no serious illnesses. Her family history was not relevant.

Examination revealed a normally developed, short (153 cm.), small (52.6 kg.) woman. She was alert and intelligent. Her skin was normal in texture and moisture. Her voice was nasal and somewhat muffled, suggesting that the use of the tongue was impaired. She made moderately visible efforts on attempting to swallow. Her eyes were slightly prominent with slight enlargement of the palpebral fissures. Eye movements were normal. The pupils were equal and reacted to light and accommodation. The ears demonstrated normal auricles, canals and drums. The hearing was normal. In the nose, the mucosa was pink and moist. The septum was not obstructive. The turbinates were normal in size and shape and were found to shrink readily. The nasopharynx was normal.

Examination of the mouth showed no questionable teeth. The gums were normal. The buccal mucosa, hard and soft palates were normal. The tonsils were out, and the fossae clean. The anterior two-thirds of the tongue were normal. At the base of the tongue, in the midline, at the region of the foramen caecum, there was a horse chestnut-sized, rubbery-feeling mass. Slightly lobulated, firm, and broadly attached to the tongue, this mass was 3 cm. in diameter. It projected above the surface of the tongue at least 2 cm. It was not movable, except by motion of the tongue itself. Its size and location clearly accounted for the patient's complaints pertaining to deglutition, respiration and speech. Movements of the tongue were normal.

The pharynx, hypopharynx and larynx were normal.

On examination of the neck, the larynx was found to be in the midline. The thyroid and cricoid cartilages were normal. The tracheal rings were easily palpated as the neck was rather thin. There was no palpable thyroid gland isthmus or lateral lobe. There was no evidence of pyramidal lobe or of thyroglossal duct. No regional cervical adenopathy was apparent. The submaxillary glands were normal in size and shape. No abnormal masses were found in palpation of the neck.

In the thorax the lungs were clear and resonant to auscultation and percussion. The heart was normal.

The abdomen showed the scar of a previous operation. There were no abnormal masses, and no tenderness or other abnormalities were noted.

The extremities were normal in appearance and in function.

The blood pressure was 100/70. Pulse and respiration were within normal limits. Urine tests were normal. The R. B. C. was 3,940,000; the Hgb., 12.6 gm.; the W. B. C., 7,900, with a normal differential. Blood Hinton was negative. N. P. N. was 25 mg./100 cc. The F. B. S. was 83 mg./100 cc. The B. M. R. was —7.

The lateral neck X-rays showed the mass at the base of the tongue, but no other abnormalities were apparent. The chest was normal by X-ray, and there was no evidence of intrathoracic thyroid.

The thyroid service at the Massachusetts General Hospital was consulted to see if radioactive iodine could be used to establish the absence of a normal thyroid gland as well as the absence of any other abnormally placed thyroid gland tissue. Dr. B. M. Dobyns and his colleagues carried out all of the work pertaining to radioactivity. They pointed out that as far as laboratory procedures go, radioactive iodine studies held a definite and unequalled place. Radioactive iodine studies made it possible to

determine whether or not the mass at the base of the tongue contained functioning thyroid tissue and, secondly, whether there was any other thyroid tissue functioning elsewhere.

The patient was given orally a drink containing 100 microcuries of radioactive iodine (131) (half life is eight days) with 10 gamma of sodium iodide as carrier. Forty-eight hours after administration, a directional scintillation counter was used to scan the patient from the area of the pharynx to the manubrium. Since the individual's fields surveyed were no larger than 2 sq. cm., an area counted laterally at the angle of the jaw coincided with the tumor mass at the base of the tongue and was found to contain the majority of the radioactivity. A survey between this point and the thyroid area revealed only a minimal amount of radioactivity apparently contributed by the area of high concentration in the tongue base. The survey over the thyroid area revealed no more radioactivity than our other control areas of the neck or thoracic cage. The conclusion was obvious that the patient had a lingual thyroid tumor and no other functioning thyroid tissue.

On the fourth hospital day, operation was performed by Dr. Moses H. Lurie and the authors. Intravenous pentothal anesthesia was employed. After cocainization of the larynx, a No. 29 Magill intratracheal tube was passed through the nose into the trachea. In the sitting-up position the hypopharynx was carefully packed with moistened gauze. The base of the tongue was exposed with a special technique. A pair of No. 2 chromic catgut sutures were passed one on either side of the midline through the tongue one inch behind the tip. Traction on these sutures enabled a second pair of sutures to be passed through the tongue one inch behind the first sutures. Traction, steady and constant, permitted a third pair of sutures one inch behind the second set. Further traction and a fourth pair of sutures provided maximum extension of the tongue. It is always remarkable how far forward the posterior third of the tongue can be brought by the above maneuver. In this case, the lingual thyroid tumor was nicely exposed. A mass the size of a golf ball was easily enucleated with scissors and blunt dissection. The posterior portion of the dissection was completed with a tonsil snare. All bleeding points were controlled. The tongue slipped back into its original position as the sutures were removed. The specimen was sent to the laboratory for special studies. The patient withstood the operation well and was returned to her bed in good condition.

The postoperative course was uneventful. For the first two days postoperatively intravenous fluids were administered as swallowing was difficult. From then on, the patient took increasing amount of fluids by mouth with less and less discomfort. Penicillin and streptomycin were given for one week. The temperature remained flat. The patient's general condition was excellent.

Forty-eight hours after operation the patient was resurveyed with the directional counter. The area of high concentration of radioactivity in the region of the base of the tongue had disappeared. There was now only a slight amount of activity in this region. This meant that a slight amount of thyroid tissue was still present and functioning at the base of the tongue.

On the eighth postoperative day the patient was discharged home. No medication of any sort was given, as it was unlikely that myxedema would develop before the next admission for recheck with radioactive iodine.

The surgical specimen removed was described grossly as spherical in shape, measuring 4.5 cm. in diameter. Small projections of muscle were adherent to one-half of the surface of the mass. The surface of the remaining half consisted of a smooth, glistening, whitish capsule. Several plaque-like masses of reddish brown tissue were present just beneath the capsule. Sectioning of the mass revealed a multiloculated cyst (see Fig. 1) containing yellowish-brown fluid. When placed in the field of the directional scintillation counter, the entire surgical specimen was shown to contain a large amount of radioactive iodine (131). No parathyroid tissue was found on close examination of the specimen.

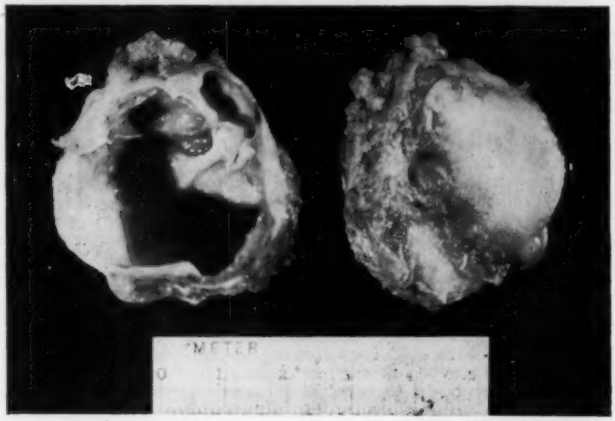

Fig. 1. Showing the multiloculated cyst in section on the left, and the protruding lingual surface on the right.

Microscopic examination showed a spherical, sharply encapsulated mass just beneath the squamous epithelium, strands of striated muscle, and mucous glands of the tongue. Although the central portion of the tissue was cystic and there were large areas of fibrosis in this region, there was a peripheral rim of thyroid tissue extending the entire circumference. For the most part the thyroid parenchyma was composed of small follicles — many of which were nonlumenated. Scattered acini were of a normal size and contained colloid. These latter were more abundant at the margin of the mass. The described follicles were distributed in a ground glass-like eosinophilic matrix characteristic of an adenoma. There were no connective tissue septa ramifying throughout the adenoma. In a few areas at the circumference of the encapsulated adenoma were islands of normal appearing follicles (see Fig. 2) which appeared to have been compressed and displaced by the enlarging adenoma. These conceivably represent remnants of normal thyroid. The radioautograph disclosed a narrow (see Fig. 3) zone of darkening around the circumference of the adenoma; this corresponded to the scattered colloid containing follicles within the adenoma and also with the previously noted foci of probably normal thyroid tissue. The cystic element of the adenoma and also the microfollicular areas, comprising the bulk of the nodule, showed no corresponding film darkening.

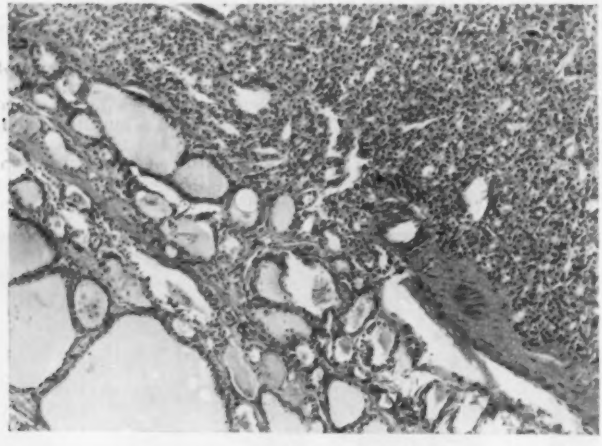

Fig. 2. Showing microfollicular adenoma at the right, above, compressing normal appearing follicles at the left, below, lying just beneath the fibrous capsule (×425).

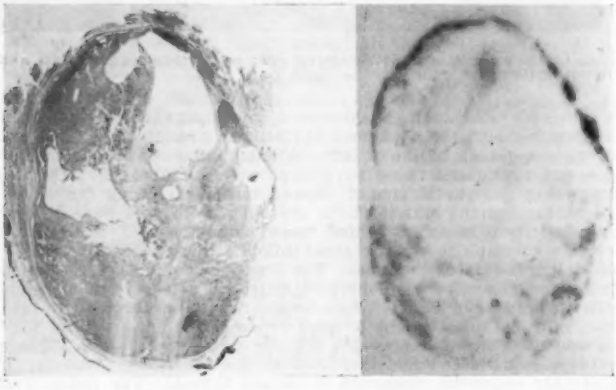

Fig. 3. Illustrating use of radioautography: the right-hand photograph shows the narrow zone of darkening around the periphery of the adenoma shown in the left photograph. In the dark a portion of unexposed film was placed over the section and it "took its own picture." The two photos are superimposable.

The diagnosis, confirmed by Dr. Austin Vickery, of the Pathology Department of the Massachusetts General Hospital,[3] was adenoma, thyroid, principally microfollicular type with central cystification and fibrosis.

On July 30, 1951, five weeks after her operation, the patient was readmitted to the Massachusetts Eye and Ear Infirmary. She had no complaints and felt only trivial twinges of discomfort on swallowing. She had gained six pounds in weight in the past month.

The physical examination was as before, except that at the operative site there was a slight hollow at the base of the tongue. Complete blood studies were normal. The urine tests were normal. The B. M. R. was —15.

Five hundred microcuries of radioactive iodine (131), with 10 gamma of sodium iodine as a carrier, were given orally in the second hospital day. Urine specimens were examined for three days. At the end of 48 hours, a Geiger counter was passed over the operated area and the entire neck. There was radioactivity demonstrable over the base of the tongue and none elsewhere, as shown by the following figures: with the background radiation count averaging 220 counts per minute subtracted from the total count over various areas of the anterior neck to be enumerated; the actual intensity of radiation over the usual thyroid area showed only 33 counts per minute, over either side of the trachea and suprasternal area, zero to four counts per minute, but at the tongue base the count rose to 1,327 per minute. The low count at the level of the cricoid was interpreted as scattering from the lingual base. Urine studies showed that 20 per cent of the radioactive iodine had been absorbed by the remaining thyroid gland tissue. Thus, it was proven that actively functioning thyroid tissue remained in the lingual area.

The patient was in excellent general condition, except for the slightly lowered B. M. R. She was discharged from the hospital on Aug. 2, 1951, on 60 mg. of thyroid daily. An appointment to return to the Out-Patient Department in two months could not be kept because the patient had moved to another state. She reported that she was symptom free and that her weight was constant. Thyroid medication was continued, and she promised to return in another two months for check-up.

DISCUSSION.

The case shows how careless doctors are at times in examining the throat. This patient repeatedly complained of dysphagia over a period of six years. At various times at least four doctors assured the patient after examination that her throat was normal. Her difficulty in breathing was sufficiently distressing so that intravenous rather than inhalation anesthesia was employed. While we may question the choice of anesthesia, it is remarkable that the posterior third of the tongue was not examined before or during the operative procedure.

The sudden onset of dysphagia six weeks prior to admission suggests the possibility of hemorrhage into a cystic cavity. The finding of yellowish-brown fluid on sectioning tends to confirm this suspicion.

The appearance and feel of the swelling at the base of the tongue preoperatively suggested thyroglossal cyst as the best diagnosis. Dr. Dobyns felt that the slight lobulation was indicative of adenoma. In that his forte is thyroid surgery, we paid little attention to his written diagnosis until the microscopic examination proved him correct. Because there was no true epithelial lined cyst, the diagnosis could not be thyroglossal cyst. It was adenoma of the thyroid with cystic degeneration.

The operative procedure was designed for the removal of a cyst rather than the complete removal of all thyroid present. The microscopic and radioactivity studies proved the correctness of this type of surgical removal. All of the encapsulated cystic adenoma was resected, leaving behind apparently normal lingual thyroid.

This is the first case at the Massachusetts Eye and Ear Infirmary in which radioactive iodine studies have been used to diagnose lingual thyroid. The fact that there was no indication on physical examination of any thyroid tissue in its usual location indicated that the mass at the base of the tongue might be thyroid, and perhaps the only thyroid the patient possessed. This situation was diagnosed conclusively by the use of radioactive iodine (131) and the very sensitive directional scintillation counter preoperatively. No other additional sites of thyroid remnants were discovered, and the conclusion was that this patient had her thyroid in the tongue and that it had never migrated caudally during her embryological period.

The use of radioactive iodine in an effort to solve a problem in embryology is unusual in that the present case presented herself 30 years after the thyroid anlage had been laid down. According to Patten, in addition to "the median bilobed primordium that arises from the floor of the pharynx at the

level of the second pouch (see Fig. 4) near the end of the fourth week, there also arise on the caudal face of the fourth pharyngeal pouches small diverticulae known by various names but more simply as postbranchial bodies. All authors agree that the diverticulae in question break loose from the fourth pouch and become incorporated with the lateral lobes

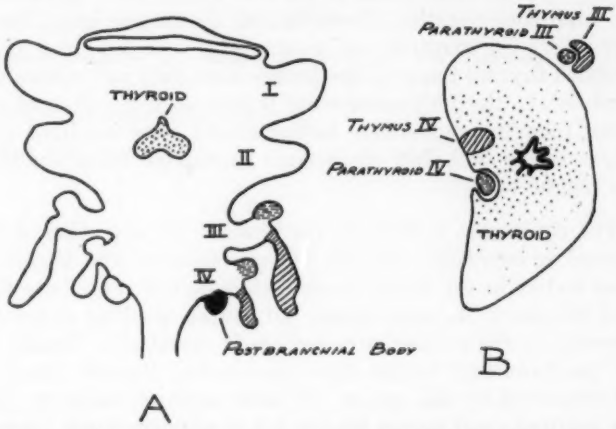

Fig. 4. (Adapted from Patten.) (A) The postbranchial body is in black and early in development is widely separated from the median thyroid primordium. (B) A later stage in development showing inclusion of the postbranchial body (black) within the migrating thyroid.

of the median thyroid primordium as it reaches posterior pharyngeal levels in its migration caudad. Once associated with the thyroid lobes, the tissue of the postbranchial origin tends to become buried in them and its subsequent history is difficult to follow. The only question is whether, in this position within the thyroid, the postbranchial tissue actually gives rise to typical thyroid follicles or merely remains as a vestigial inclusion of unknown significance."*

The present case seemed to us to be an ideal "critical experiment" to find out exactly what does become of the postbranchial bodies and if they really do form thyroid tissue. The use of radioactive iodine was of decisive help in clarifi-

*Bradley M. Patten: Human Embryology, 1948, pp. 530-531.

cation of this point as it localizes in thyroid tissue wherever it may have formed or migrated. The first administration of radioactive iodine and the subsequent localizing by use of the directional scintillation counter revealed that the only radioactivity was at the base of the tongue, there being little or no radioactivity along the usual course of the migrating gland and none in the upper thorax. The possibility still remained, however, that other sites of thyroid tissue were masked by the high concentration of activity at the tongue base; therefore, a second counting one month later was done and again revealed thyroid tissue in the tongue base *only* and in no other location. If the ultimobranchial bodies do form thyroid follicles, then the radioactive iodine should have localized elsewhere in the neck, but our patient showed no other site than the tongue base.

The conclusion is that, in the absence of migration of the thyroid primordium and the incorporation of the postbranchial bodies in the gland, these bodies do not contribute thyroid follicles to the adult gland. Of course, whether or not the presence of the median primordium is essential to transform the postbranchial bodies into functioning thyroid tissue is not answered by this paper. It seems definite, however, that the postbranchial bodies do not by themselves form thyroid tissue.

CONCLUSIONS.

1. With a history of dysphagia and dyspnea, it is amazing that this patient could go through an operation under general anesthesia and be cared for repeatedly during five years by physicians without having her hypopharynx examined.

2. When a tumor involves the lingual thyroid area, radioactivity studies should be done to determine the exact location and function of thyroid tissue in the patient.

3. Suture traction of the tongue provides an excellent exposure of the posterior third of the tongue for the surgical removal of tumors or cysts.

4. Since cystic degeneration commonly occurs in adenomas of thyroid tissue, surgical removal is preferable to medical treatment.

5. The distribution of radioactivity in this patient throws new light on the embryological development of the thyroid gland.

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THE IMPORTANCE OF NUTRITIONAL SURVEYS IN OTORHINOLARYNGOLOGIC PRACTICE.*

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The patients that present themselves to otorhinolaryngologists with symptoms and complaints referable to the ear, nose and throat fall principally into three general classifications: first, those with a purely local disease, local in origin and largely local in manifestations; second, those with local manifestations of diseases that are generalized in character or distant in origin, and the third class is a combination of the first two groups.

Since nutritional failure is a generalized condition with many and varied localized manifestations, it is the purpose of this paper to study those manifestations that affect the field of otorhinolaryngology and more especially to evaluate the methods for nutritional study that may efficiently and conveniently be used in everyday clinical practice.

INCIDENCE OF NUTRITIVE FAILURE.

The incidence of nutritive deficiency varies in different population groups and varies also with the criteria used for measuring deficiencies. Moose,[1] citing a relatively recent National Research Council report, states that about two-thirds of the American public is suffering from a nutritional deficiency. Biskind and Williams[2] surveyed 183 boys in a children's institution and found that 90 per cent had glossitis; 67 per cent, gingivitis; 19 per cent suffered from cheilosis; and 60 per cent had symptoms of avitaminosis related to the nervous system. Pierce[3] and his associates found evidences of inadequate nutrition in 85 per cent of a group

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of Vermont school children. Adamson *et al.*[4] very carefully studied 868 representative residents of Newfoundland and noted signs of malnutrition in a large percentage. Thickened conjunctiva was present in over 76 per cent; cheilosis in 68 per cent; and active severe dental caries in 61 per cent. Other signs were present in lesser proportions. In another study, also in Newfoundland, McDevitt and associates[5] studied seasonal variations in blood values of vitamin C. Many were found deficient, especially among the mothers. The main clinical lesions were gingivitis, dental caries and hyperkeratosis. Out of 284 patients studied carefully by clinical histories and physical examinations, they found that 233, or 82 per cent, showed deficiency in at least one of the vitamin B components. Most individuals presented multiple deficiencies. Kruse[6] studied 143 persons in a low income group and found gross evidence of xerosis in 45 per cent and microscopic evidence of xerosis conjunctivae in an additional 54 per cent (total, 99 per cent).

Perhaps one of the best and most recent surveys is that of Spies,[7] who carefully examined 10,851 persons. Nutritive failure was found in over half of the individuals; namely, 5,711 cases. It becomes readily apparent that well over half of the American public suffers from varying degrees of nutritional failure and we may assume that among patient groups who seek medical advice, because of various complaints, the proportion must be still greater. Thus, the average physician who, on any given day, sees 30 average patients in his consultation rooms can be quite certain that 15 to 20 of those patients would undoubtedly be benefited by nutritional investigation and guidance. Statistics of group studies from other parts of the world bear a similar relationship. A particularly informative paper has been published by Gillman and Gillman[8] on the problem as it affects Negroes and whites in South Africa.

THE NATURE OF NUTRITIVE FAILURE.

It is not within the scope of this paper to review in detail all of the signs and symptoms of nutritive failure associated

with the respective nutrients. Standard reference works by such authors as Youmans[9] and McLester[10] quite adequately portray that part of the problem. Most writers on the subject agree that rarely is there a nutritional deficiency of only one or two nutrients. Most deficiencies involve multiple nutrient factors.

One of the earliest signs of nutritional failure is fatigue, in its varied forms. Associated with this may be various feelings of nervous tension. Almost any digestive disturbance can be a symptom of nutritional failure as is a host of dermatologic conditions. More specifically, in the field of otorhinolaryngology we should suspect the possibility of nutritional irregularities if there is evidence of dental or gingival disturbances; soreness, fissuring and atrophy of the tongue and lips; sinusitis, postnasal drip, nasal or other allergy; chronic pharyngitis and sometimes dysphagia. Even the aural system may reveal nutritional deficiencies and in our neighboring field of the eye there are often signs of vitamin A and riboflavin deficiency.

HISTORICAL.

Recently, Strauss[11] reviewed the otolaryngologic literature available in the year 1850. Among other items he found that a nourishing diet was advised by one author for otitis media. Another writer reported the "cure" of a facial and orbital malignant tumor by a bread and milk diet. (Incidentally, a Boston newspaper records the obituary of the "cured" patient six months later, the cause of death not being stated.) When comparing this paucity of nutritional interest in our field with the many excellent factual articles being written each year at the present time, we can see that real progress is being made in the field of nutrition as it relates to otolaryngology.

Furstenberg,[12] in 1949, chronicled 100 years of otolaryngology and a perusal of his paper reveals little or no reference to nutrition until the end of the paper when he mentions amino acid therapy in surgical problems. Nevertheless, the field of nutrition was being covered by enthusiastic pioneers.

J. A. Stucky,[13] early in this century, pleaded for nutritional studies to be made in rhinologic patients. His disciple, D. C. Jarvis,[14] later carried the torch, and Eugene R. Lewis[15] likewise was an early advocate of nutritional evaluation in our field of practice.

In more recent years the contributions of Roberts[16] and Moose[1] and the yearly contributions by Isaac Jones[17] and his associates stand out as milestones of progress in otolaryngologic nutrition.

Excellent reviews have also been given by Perlman,[18] Cody,[19] Wallner,[20] Beck[21] and Coakley.[22] Even the foreign literature[23] has made excellent contributions. Many of the early papers on otolaryngology and nutrition contained much conjecture, and the conclusions were often based upon circumstantial evidence; however, as the field of nutrition in general has become clarified from year to year, the ideas and concepts as they pertain to otorhinolaryngology have also begun to crystallize into more factual patterns.

COMMON LESIONS.

As stated previously, rarely do we find nutritional deficiencies due to the lack of a single food factor. More commonly, many factors are involved; however, there are certain lesions in our anatomical field that seem to represpent to a large extent specific deficiencies. Seborrheic lesions about the eyes, ears and nares may be associated with a riboflavin deficiency, as evidenced by improvement under specific therapy. The fissures at the corner of the mouth, irritated lips and cheilosis are also evidences of a riboflavin lack. Atrophy of the tongue papillae, redness and soreness, and fissuring of the tongue are usually associated with B complex deficiencies, especially niacin. These lesions are commonly seen in so-called pernicious anemia, sprue, and in certain cases of diabetes. Martin and Koop[24] have published an excellent paper on the "Precancerous Mouth Lesions of Avitaminosis B." This should be read by every practitioner who looks into a patient's mouth. In their work at the Memorial Hospital in New York they have

clearly demonstrated that the degenerative mucous membrane lesions in and about the mouth produced by avitaminosis B, very definitely favor the subsequent development of cancer on the site of these lesions.

Dental caries is a very definite evidence of nutritional failure, past or present or both. The monumental work of Price[25] has clearly settled this question. Gingivitis, with or without bleeding, suggests ascorbic acid and calcium deficiencies.

Roberts[16] states that hypertrophied lymphoid follicles in the pharynx can represent an iodine deficiency. There are some workers who feel that such hypertrophies may also be associated with an insufficient intake of unsaturated fatty acids or so-called vitamin F. Goodale[26] has reported a case of laryngitis apparently due to B complex vitamin deficiency. The patient also had a sore tongue and esophageal distress. Personally, I have observed two cases with esophageal pain and distress in which esophagoscopic examination revealed only a smooth atrophic lining resembling somewhat the smooth tongue of vitamin B deficiency. Both patients had definite evidence of vitamin B deficiency in their diet. One was relieved by appropriate dietary guidance. The other patient was an alcoholic and lived at a great distance and has not been observed again. His mouth and lips presented typical evidences of riboflavin and niacin deficiencies.

Jarvis[14] postulated a nasal syndrome due largely to the use of an excess of acid ash food in the diet. He stated that a pale nasal septum mucosa indicated too much acid and a more red mucous membrane a more alkaline dietary intake. He reported relief of nasal symptoms by changing the diet from acid ash to alkaline ash. In the light of more recent nutritional knowledge, one is led to wonder if the improvement obtained was purely one of acid versus base, or whether the basic foods did not supply certain other beneficial nutrients that had no direct acid-base relationship.

Roberts[16] has proposed a nutritional failure syndrome based upon six main points, namely:

1. Postnasal mucus drip without sinus infection.
2. Sensation of nasal stuffiness without organic obstruction.
3. Sinus pain without sinus infection.
4. Fatigue without organic findings.
5. Neuritis.
6. Allergic manifestations.

Every rhinologist has seen many of these unhappy patients and some of us, due to our ignorance many years ago, may have contributed to their unhappiness by performing unwise operations upon them. In more recent years our surgical conservatism has increased and we are now able to help these patients by nutritional therapy and guidance after first ascertaining the cause of the nutritional failure.

In the past 20 years much interest has developed in the nutritional approach to deafness and vertigo. Guggenheim[27] obtained some encouraging results in vitamin and mineral therapy of otosclerosis. Covell[28] studied the otic capsule in avitaminotic animals and described various bone and soft tissue lesions, but could report no definite vitamin therapy approach to ear disease. Mellanby[29] studied puppies with avitaminosis A and noted serous labyrinthitis as well as bony lesions in the vicinity of the cochlear and vestibular nerves. Perlman[30] in his studies of young rabbits was able to produce bone lesions when vitamin A was lacking, but no cochlear impairment could be shown. He advises caution in concluding that avitaminosis A is important in deafness. Lawrence[31] was unable to confirm the histologic findings of Mellanby[29] and found no behavioral responses indicating hearing loss in vitamin A deficient rabbits. More recently, Lobel,[32] Anderson *et al.*[33] and Bau and Savitt[34] have reported encouraging results in progressive deafness and tinnitus with massive doses of parenterally administered vitamin A. Further reports on this work may prove to be interesting.

Atkinson[35] has reported on the use of large doses of niacin and riboflavin in Ménière's disease and feels that deficiency

of the two factors plays an important rôle in the etiology. In this connection it should be pointed out, however, that in the work of Atkinson[35] and Lobel[32] the dosages of vitamins used are far in excess of any conceivable physiologic requirements for those vitamins. Their therapeutic effect, hence, may in part be pharmacologic instead of altogether nutritional. For example, the vasodilating effect of niacin.

During World War II, DeRaadt[23j] studied Allied prisoners of war in Japanese camps in the Far East. He found 160 patients with otoneurologic syndromes due to malnutrition. Many were pellagrins and he suggests that Ménière's syndrome may be associated with "monosymptomatic" pellagra of an early stage. Though a niacin deficiency is the prominent factor of pellagra, he stresses that it is actually a deficiency of multiple factors. A riboflavin lack was deemed to be largely involved in bringing out central nervous system lesions. The work of Atkinson[35] takes on added interest in view of the findings of DeRaadt.[23j]

Selfridge[36] had previously reported on the use of vitamins in deafness, and more recently he reviewed the nutritional and endocrine factors in Ménière's disease. He felt that electrolytes, vitamins and endocrine secretions play an important rôle in the etiology. In the neighboring field of ophthalmology we naturally think of xerosis ophthalmia and night blindness as frequent evidences of vitamin A deficiency. Corneal vascularization may indicate riboflavin deficiency, but this is not an infallible test, as pointed out by Lyle *et al.*[37]

Elderly people have been shown by Rafsky and Newman[38] to give frequent evidence of subclinical deficiency disease. In 38 carefully studied, apparently normal aged individuals they found vitamin deficiencies in 75 per cent. My personal experience indicates that this percentage would be quite representative in my own practice.

During the past few years much interest has centered about the hypoprothrombinemia which is so often produced after tonsillectomy by the use of acetylsalicylic acid, used for the relief of pain. This condition can be relieved or largely pre-

vented by the pre- and postoperative use of vitamin K.[39] Fox and West[40] feel that in addition to the vitamin K deficiency in postoperative hemorrhage cases there may be a protein deficiency. They advocate a preoperative study in each patient to include a search for constitutional and dietary disorders.

The rôle of nutrition in surgical patients has in recent years received much deserved attention. For the healing of soft tissue wounds adequate protein, ascorbic acid and other nutrients are essential. Bone wounds need calcium, phosphorus and vitamins B, C and D in particular.

Elman[41] has pointed out that protein deficiencies are actually prevalent in clinical medical and surgical practice, and that the average physician often fails to recognize this fact. If the patient is temporarily unable to ingest, digest or absorb protein foods, amino acid therapy should be given orally or parenterally as indicated. An excellent bibliography and literature abstract booklet is available on the subject of "Proteins in Relation to Surgery,"[42]

Holman[43] studied the incidence of dietary deficiency in 70 patients from the low economic levels admitted to surgical wards for surgery. He found that 44 per cent of the patients had lower than normal vitamin C intakes. An additional 13 per cent were found to be on the verge of clinical scurvy.

NUTRITION AND ALLERGY.

A patient who is allergic to foods may present a serious nutritional problem if the allergenic foods are important staple foods or are many in number. The avoidance of many of these staples may actually aggravate the allergy because of the secondary malnutrition. As otorhinolaryngologists we see many of these individuals and it is our duty to assist these patients in their nutritional rehabilitation. It is not sufficient to limit ourselves to multitudinous skin tests and to the administration of desensitizing antigens or vaccines. Such an approach to the allergy problem does not uncover or relieve certain of the underlying causes of the allergic state which must obviously be metabolic derangements, part of which are often nutritional. The concept that a nutritional deficiency

might be a basic cause for an allergic state is not altogether new, for Russell Wilder[44] has stated, "Yet some evidence suggests that allergy itself may be occasioned by unbalanced diets."

Oelgoetz[45] and associates, in an excellent paper published in 1934, postulated, and seemed to have shown, that normal individuals have adequate amounts of pancreatic enzymes circulating in the blood stream to digest various foreign proteins and incompletely digested proteins which might gain entrance into the blood stream. Allergic individuals usually have a deficiency of these circulating enzymes. I have personally verified this latter fact in my own laboratory on many occasions. Consequently, Oelgoetz[45] points out that individuals with low blood enzyme titers who might eat too much at one time, place such a great load upon the digestive system that much incompletely digested protein may be absorbed, with the result than an acute allergic reaction ensues. Such patients need careful adjustment of their eating and living habits to avoid the sudden strains on their weakened enzyme system. I know that others besides myself have had the gratifying experience of seeing marked improvement or remissions in allergic individuals by merely correcting their eating habits and assuring a full nutrient supply without the necessity of avoiding certain foods and without desensitization programs. Dorothy Clark[46] has recently published an excellent paper on the treatment of allergic conditions from the endocrine standpoint, following some of the concepts put forth by Oelgoetz.[45] Other excellent papers in this same general field, dealing with proteins in allergy are those of Adams[47] and Vermilye and Thompson.[48] They point out that allergic patients often fail to digest and utilize protein foods and they are often benefited by protein hydrolysates. In my clinical practice I have found this to be true, and I am certain that the metabolic approach to the allergy problem will eventually overshadow the immunologic approach. The recent success with Cortisone in asthma is an encouraging milestone of metabolic progress, though it is not the final solution to the asthma problem.

NUTRITION AND THE ENDOCRINE SECRETIONS.

In their action in the body, both vitamins and hormones act as catalysts in the various metabolic reactions. Whereas the former are largely obtained from without the body in our daily food, the latter are largely elaborated within the body. Accordingly, the two classes must be considered together, and no practice of nutrition is adequate without some consideration of the related hormones.

Sieve[49] has published an excellent series of papers on the subject in which he shows the very important and intimate interrelationship between vitamins and hormones. Jungck and Heller[50] have also provided a most useful summary of the more common features of nutrition in endocrinology. For example, androgenic failure may produce a lack of mineral retention and will make nutritional correction difficult if the androgen failure is not compensated. They state, "One may infer . . . that the hormones increase the efficiency of utilization of proteins and minerals, and that in the absence of the hormones, the nutrients, if given in large enough quantity, can (possibly by the law of mass action) in part overcome the hormonal deficiency insofar as the metabolic defects of such a deficiency are concerned." Many patients with impaired nutrition are unable to handle large quantities of foods. Consequently, we cannot depend for results upon excessive and massive doses of nutrients in these patients, but must secure optimum utilization of such nutrients as proteins and certain minerals by the use of androgens or other hormones appropriate to the individual patient.

The dependence of the thyroid hormone upon adequate food iodine is, of course, an old and classic example of the problem at hand, but as otolaryngologists our endocrine approach must extend beyond the dispensing of thyroid hormones.

METHODS OF STUDY.

There are at least seven methods by which we obtain information relative to the nutritional state of our patients. They may be classified as follows:

1. Patient's complaints. These are symptoms or signs that the patient volunteers as he brings his complaints to the physician. For example, a sore tongue, or bleeding gums. These would suggest nutritional failure.

2. Further symptoms of nutritional failure not complained of by the patient, but elicited in the nutritional history. For example, a patient with a sore tongue may not think to volunteer to a laryngologist that he also has excessively soft or liquid stools, as in sprue. (The method of eliciting a nutritional history will be given below.)

3. Living habits, diseases or drugs which either alter the patient's ability to digest and assimilate nutrients or which produce an added requirement for certain food factors. Examples are: no breakfast, light lunch and a heavy evening meal. Obviously such a patient cannot maintain optimum nutrition. Patients with achlorhydria or "colitis" obviously do not utilize their foods to the optimum. Users of mineral oil may lose many fat soluble vitamins.

Nutritional therapy will often result in failure if living habits which prevent optimum food utilization are not ascertained and corrected. Too great stress cannot be laid upon this point for it is very important.

4. The diet eaten. This is elicited in the nutritional history and its sufficiency or deficiency determined.

5. Physical examination for evidences of malnutrition. Otolaryngologists should make at least a limited number of observations outside of the ears, nose and throat. For example, weight, blood pressure, skin, hands, hair, eyes, etc.

6. Laboratory tests. For example, allergic individuals often show a lowered pancreatic function and sometimes increased serum potassium. A patient may have hyperinsulinism. A glucose tolerance test will be decisive and the therapy consists, in most instances, of a simple regulation of the patient's eating habits.

7. Therapeutic test. Occasionally verification of our tentative diagnoses must await the therapeutic test.

THE NUTRITIONAL HISTORY.

The taking of an adequate nutritional history can be a very time-consuming procedure and is practically an impossibility in the average clinical practice, if the physician elicits it orally himself. In large clinics trained technicians might do the work; however, if a questionnaire type of history is used, much time can be saved by the busy practitioner as the patient records the history at home. Various history forms and outlines have been recommended by Moose,[1] Roberts,[16] Levinton,[51] Spies[7] and Burke.[52]

Obviously, any dietary history can suffer from inadequacies, but it is still a very useful tool, as pointed out by Burke,[52] and it does fill a need. Some nutritionists try to overcome the deficiencies of a history by obtaining an inventory of all foods eaten for two weeks and then tabulating the amounts of the various nutrients, minerals, etc., as derived from food tables. This method, too, leaves room for possible error. As in any history, the patient's veracity is involved. Then comes the question of the quality of the food; where it was grown; how it was harvested and shipped; how it was prepared, etc. Finally, after the patient ate the food, did he utilize it fully? Burke[52] reported no consistent success in obtaining seven-day menus from patients, and she recommends that only a one-day menu be recorded.

The nutritional history form which I use is patterned somewhat after that of Roberts[16] with various modifications. Two pages are devoted to the several groups of foods and two pages to living habits and symptoms which might pertain to the patient's nutrition. As a check against the patient's estimate of his dietary habits he records his entire menu for two days. This is a very valuable feature.

Simple language is used so that any patient who can read can use the history. As can be seen (see Fig. 1) from the reproduced history it is crowded together to conserve space in filing, etc. On the occasion of the patient's first visit he receives the history with brief instructions on the importance of the form and its use. The form is executed at home and

consumes at least an hour of time in the average case. It is then mailed to the office or brought back on the occasion of the second office visit.

FIG. 1, PAGE 1.

NUTRITIONAL HISTORY

INSTRUCTIONS: Approximately how many EACH WEEK of the following articles of food do you consume? When possible, put FIGURES in blank spaces. If a food is taken only occasionally, write OCC, in the blank space after the food. If a food is not taken, write NONE in the blank space after the food.

1. Glasses of sweet milk....., Glasses of Buttermilk....., Servings of cheese.....

Ice Cream....., Soy Milk....., Soy Cheese.....

2. Eggs....., Strips of bacon....., Servings of meat....., Servings of fowl.....,

Servings of fish....., Canned meat substitutes.....

3. Servings or portions of Pie....., Cake....., Candy....., Cookies....., Doughnuts.....,

Jello....., Other desserts most commonly eaten.....

Cooked cereal....., Dry cereal....., Rice....., Spaghetti or Macaroni.....,

Pan Cakes....., Waffles....., Crackers.....

4. Servings of vegetables: White Potatoes....., Mashed Potatoes....., Sweet Potatoes.....,

Corn....., Carrots....., String Beans....., Green Peas....., Pumpkin....., Squash.....,

Spinach....., Chard....., Celery....., Lettuce....., Tomatoes....., Radishes.....,

Broccoli....., Cauliflower....., Cabbage....., Brussels Sprouts....., Asparagus.....,

Onions....., Beans (dried)....., Soy Beans....., Lentils....., Garbanzos.....

5. Servings of fruit: Oranges....., Grapefruit....., Pineapple....., Apples.....,

Bananas....., Prunes....., Dates....., Raisins....., Figs....., Melons.....,

Strawberries....., Berries (other)....., Cherries....., Grapes....., Fresh

Peaches....., Fresh Pears....., Apricots....., Plums....., Apple Sauce.....,

No. of Olives....., Olive Oil (teaspoon)....., Avocados....., Canned Fruits (kinds).....

6. Peanuts....., Peanut Butter....., Pecans....., Walnuts....., Almonds.....,

Filberts....., Others.....

7. Marmalade....., Jam....., Jelly....., Sweet Pickles....., Sour Pickles.....,

Honey....., Candy bars....., Sticks of Gum.....

8. Glasses of Orange Juice....., Grapefruit Juice....., Pineapple Juice..... Apple Juice.....

Lemonade....., Grape Juice....., Cranberry Juice....., Tomato Juice....., Coca-Cola.....

Bottles of Soda Pop....., Glasses of Ginger Ale....., Other Soda Fountain drinks.....

The questions and items are grouped in such a manner that the physician with a little practice can analyze the chart, detect the important points, and mark these with a red pencil, in a total of about three minutes in the average case.

FIG. 1, PAGE 2.

PER DAY—How Many?

1. Portions of butter (½ inch x 1 inch x 1 inch)....., Margarine.....

2. Portions of white bread (portion is one slice, one roll, etc.)....., Whole wheat bread.....,

Rye bread....., Corn meal bread....., Oatmeal bread....., Soy bean bread.....,

Baking powder biscuits....., Sweet rolls.....

3. Glasses of water....., of alcoholic beverages and what kind.....

4. Cups of coffee....., Kaffee Hag or Sanka Coffee....., Postum....., Ovaltine.....,

Cups of Tea....., Cocoa.....

5. Do you use table salt sparingly....., freely....., moderately.....

6. Do you use pepper sparingly....., freely....., moderately.....

7. Do you use vinegar sparingly....., freely....., moderately.....

8. Catsup and spicy sauces sparingly....., freely....., moderately.....

9. If you add white sugar to tea, coffee or other foods at the table, how many teaspoonfuls each day?.....

10. Is this your average diet for the past three or four years? .....

11. What foods or food combinations, if any, disagree with you?.....

.....

12. Are you allergic to any foods?.....

13. Fond of meats? (Draw a line through the *incorrect* answer) (yes-no), Fats? (yes-no), Sweets? (yes-no), Vegetables? (yes-no), Fried Foods? (yes-no), Chocolate? (yes-no).

14. Make a list of what you had for breakfast, lunch, and supper for two representative days such as:

	Today:	Yesterday:
Breakfast	.....	.....
	.....	.....
	.....	.....
Lunch	.....	.....
	.....	.....
	.....	.....
Supper	.....	.....
	.....	.....
	.....	.....

Then follows the consultation with the patient in which the pertinent facts of the history are discussed, together with any correlated physical and laboratory findings. If some features

FIG. 1, PAGE 3.

PERSONAL REPORT

INSTRUCTIONS: In answering questions draw a line through the YES or NO that does NOT represent the correct answer. If you wish the answer to read YES, draw a line through NO. If in doubt, say "NO." In blank spaces write in appropriate answer.

Do you sleep soundly? (yes-no) How many hours generally? .....
Do you have difficulty getting to sleep? (yes-no)

At what hour do you usually retire?..... Get up?.....
Do you tire easily? (yes-no) Do you have a sense of exhaustion? (yes-no)

Do you smoke? (yes-no) How many each day? Cigars....., Cigarettes....., Pipe.....

How many bowel movements do you have per day?.....

Do you ever take medicine for your bowels? (yes-no) How often? .....

What do you usually take?.....Are you taking any vitamin now? (yes-no)

Are you using nose drops now? (yes-no) What kind? .....

Do you use Aspirin or other pain remedies? (yes-no) What and how much?.....

Are you taking any other medicine? (yes-no) State what, how often, how long, and how much.....

..... Allergic to any?.....

What is your usual hour for Breakfast?..... Dinner?..... Supper?.....

Which is your heaviest meal?..... Do you eat "snacks" between meals or at bedtime? (yes-no)
Is your appetite good? (yes-no) Are you afraid of certain foods? (yes-no) Do you crave sweets? (yes-no)
Sour foods? (yes-no) Have you ever been on a "special" diet? (yes-no)
Have you ever had ulcer of stomach? (yes-no) Do you have colitis? (yes-no) Diarrhea? (yes-no)
Do you have gas or burp after eating? (yes-no) Are you subject to nausea or vomiting? (yes-no)

Are you subject to headaches? (yes-no) Location?.....
Have you any hearing impairment? (yes-no) Do you have noises in your ears? (yes-no) Discharging ears? (yes-no)
Are you ever dizzy? (yes-no) Eyes burn or feel tired after moderate use? (yes-no) Eyes feel rough? (yes-no)
Do bright lights hurt your eyes? (yes-no) Do you see poorly at night? (yes-no)
Does your hair fall out faster than it should? (yes-no)
Do you have much sneezing? (yes-no) Do you have hay fever? (yes-no) Wheezing? (yes-no)
Allergy? (yes-no) Hives? (yes-no) Does your skin itch? (yes-no) Ears? (yes-no) Nose? (yes-no)

If allergic, what substances bother you?.....

Is your skin dry or moist?..... Does your hair tend to be dry or oily?.....
Do you have brown spots on skin of neck or back of hands? (yes-no) How long?.....
Are your hands and feet cold? (yes-no) Do you feel weak or strong generally?..... Do you tire easily? (yes-no)
Do you take cold easily? (yes-no) Have sore throat often? (yes-no) Clear your throat often? (yes-no)
Do you have an unpleasant taste in your mouth? (yes-no) Bad breath? (yes-no)
Do you have difficulty breathing through your nose? (yes-no)

Do you have mucus dropping in your throat? (yes-no) Is it slight, moderate, or severe?..... What is its usual color?..... Do you have sinus trouble? (yes-no)
Do you have a choking sensation in your throat? (yes-no)
Do your teeth decay easily? (yes-no) Do your gums bleed when they are brushed? (yes-no)
Are your lips ever sore? (yes-no) Sore tongue? (yes-no) Burning tongue? (yes-no)

FIG. 1, PAGE 4.

Do you have rheumatism? (yes-no) Neuritis? (yes-no) Do you have muscle cramps any place in body? (yes-no)
Do you ever have pain or tight feeling in your chest or in the region of your heart? (yes-no) Do you have any heart symptoms? (yes-no) Are you short of breath on mild exertion? (yes-no)
Do your finger nails bend, break or tear? (yes-no)
Have you recently lost weight? (yes-no) Gained? (yes-no) What is your present weight?.....

Has there been diabetes, tuberculosis, cancer, asthma, hay fever, or deafness in your family? Name which.....

During cold weather do you sleep with the window open at night? (yes-no) Do you perspire freely? (yes-no)

How many times do you pass water during the day?..... During the night?.....
Do your lower limbs ever swell? (yes-no) Do your hands and feet go to sleep easily? (yes-no)
Are you nervous? (yes-no) Difficult to relax? (yes-no) Have you ever had a nervous breakdown? (yes-no) Are you tense or high strung? (yes-no)
Are you inclined to worry? (yes-no) Feel sluggish mentally? (yes-no) Poor memory? (yes-no)
Was your childhood a happy one? (yes-no) Are you personally happy now? (yes-no)

What are your hobbies? .....

What type of reading do you prefer? .....

What type of amusements do you prefer? .....

What is your occupation? ..... Is your work strenuous? (yes-no) .....

What out-of-doors physical exercise do you get? .....

How many miles do you walk per day?..... Do you have a vegetable garden? (yes-no)

What serious diseases have you had? .....

.....

What operations? .....

.....

Have you ever been anemic? (yes-no) What laboratory tests have you had during the past year?.....

.....

What symptoms do you consider most important in your case? .....

.....

Name ..... (Please sign)

DO NOT WRITE IN SPACE BELOW

need further elucidation the physician can quickly ask the necessary questions, and if further laboratory studies are needed these can now be planned.

On the basis of the above findings most of the necessary nutritional guidance can be given on the occasion of the second office visit. It is not my practice to use printed stock diets. Most instructions are given orally and an attempt is made to build the new diet as closely as possible around the patient's old diet, based upon his racial, cultural and family background. Our percentage of success in dietotherapy will be considerably lowered if we make routine, sweeping, radical changes in the diets of our patients. Most alterations of that type are unpleasant to the average ambulatory individual. Occasionally it is of course necessary to make drastic changes.

A recent interesting report on the value of nutritional therapy has been provided by Rawlings.[53] Among 315 children, 125, or 39.5 per cent, gave evidence of present impaired nutrition; 227 children had not had previous tonsil and adenoid surgery and of these, 139 were found to be in need of such surgery. All 227 children were placed on vitamin and mineral supplementation therapy as seemed indicated in each case. In three to 32 months all but six of the 139 children had been improved to the point where tonsil and adenoid surgery were deemed to be not necessary. Not only was infection lessened by nutritional therapy, but the children developed better appetites, gained in weight, increased growth, improved in their school work and evidenced better attitude and behavior patterns.

The physician who attempts to give nutritional advice should be well versed in standard texts such as the American Medical Association Handbook of Nutrition,[54] Youmans,[9] McLester,[10] Wohl[44] and Sherman.[55] An excellent monthly review is also available for the busy practioner.[56] Most patients are deeply grateful for sound nutritional advice; they appreciate knowing why certain habits are helpful or harmful. A few minutes of time spent in explaining in simple language a bit of body chemistry and physiology creates a desire in the patient to adjust his living program so that health can reasonably be expected. In our nutritional counseling we are not

so much interested in prescribing exact amounts of specific nutrients as we are in instructing the patient concerning the dietary principles he should follow in improving his diet.

Our therapy in nutrition must be based upon an adequate history. We would deem it ridiculous to prescribe drugs without bothering to take a clinical history. Might we not ask if it is not equally ridiculous to hand out (prescribe) printed diet sheets for certain conditions without first obtaining a nutritional history? Spies[7] has aptly said: ". . . It is deemed unwise for the physician to approach any patient without a complete study on which he can base his therapy. . . . " Nutritional therapy is sometimes dramatic and may occasionally produce quick results. On the other hand, in the words of Spies:[7] "It must be kept in mind, however, that nutritive failure usually has taken months or years to develop and it cannot be overcome quickly."

There may be some otolaryngologists who would prefer to refer their patients to a nutritionist for therapy. For such the nutritional history form would still be very useful as a screening device to determine which patients should be referred.

ANAYSIS OF SERIES.

I have recently evaluated 500 nutritional histories obtained in my private practice of otorhinolaryngology. Analyses of consecutive segmental groups of the histories in the series of 500 showed such a close similarity in percentage findings in each group that it became clear that no substantially greater value could be gained from a larger series, even though more histories were available for analysis. It would require a substantial book if we were to tabulate each question used in the questionnaire, so only a few salient points will be discussed herein.

Calories. In an ambulatory clinical practice I have not found much necessity for counting calories as a routine measure. The quality of food and how it is utilized are more important than the absolute quantity of food ingested. A rather

quick glance at the diet history will suggest whether or not the total calories are correct. The handiest index of adequacy of calorie consumption is of course the body weight. Normal caloric requirements are modified in any given case by such factors as age, sex, skeletal size, body metabolism, occupation, climate, disease, etc.

Protein. Only 13 per cent of our series of 500 were deemed to have inadequate protein intakes. While we did not weigh and count the calories, the obvious deficiencies recorded against these patients were such that guidance as to protein intake was imperative for those individuals. In addition there were other patients who could well afford some advice on proteins to improve the quality and variety of their fare in order to assure a "safe" margin above their minimal needs.

Carbohydrates. Eighty-one per cent of the series ingested what could reasonably be considered an excess of refined carbohydrates, *i.e.*, refined sugar and white flour products. Cowgill,[57] in 1939, estimated that about one-half of the average American's diet (caloric value) consisted of white flour and refined sugar. When one considers how much of these two substances is hidden in many of our supposedly good foods the figures are not difficult to believe. In some urban centers the percentage is undoubtedly higher than 50 per cent. The reader may ask what standard I used as *excess* in this series. Again I must state that the food was not weighed, but to illustrate: we would regard a weekly diet with several helpings of ice cream, several of pie and cake, doughnuts, candy or gum daily, "soda pop" several times weekly, the exclusive use of white bread, and daily sugar in the coffee and on the cereal as hardly a wholesome program. Undoubtedly the excessive use of refined carbohydrates is the major nutritional error in America today. So-called uncivilized tribes, when they begin to use our carbohydrate delicacies, soon acquire some of our "civilized" diseases such as dental caries (see Price[25]). In advising our patients of their nutritional status we endeavor to explain why such things as excessive amounts of refined carbohydrates are injurious. We explain their local

action in the mouth and digestive tract, and then their systemic action in depleting the B vitamins in the course of their metabolism.

Fruits and vegetables. These were grouped together for convenience. Thirty-five per cent of our patients had such a poor total amount or variety of these foods as to be considered below "average." Too little fruit seemed to be a more common error than too few vegetables, and had we analyzed the fruit intake separately a larger percentage would no doubt have been obtained. In our geographic location many individuals seem largely to satisfy their fruit menu with citrus juices. This is a serious error. There are many sufferers in our vicinity with perennial hay fever or allergic rhinitis. Some of these individuals, though not sensitive to oranges *per se*, will get allergic flare-ups if they drink heavily of orange juice. A probable explanation is that the readily available high potassium content of the oranges further upsets the potassium-sodium ratio in the blood, which is already leaning too heavily to the potassium side.

Breakfast. Eighty-four per cent of our patients did not eat an adequate breakfast. Some ate no breakfast, others merely a hasty snack, and others a breakfast too stereotyped, or lacking in good quality proteins, fruits and vitamins. The importance of this problem has been recognized in recent studies. Jackson and Schuck[58] found inadequate breakfasts in university women and on checking grade school children, ages nine to 14, they found them likewise failing to eat adequate breakfasts. Of 1,300 children studied, only 4 per cent could be said to have had adequate breakfasts from a nutritional point of view.

Gallaway and Robertson[59] studied school children in Toronto in cooperation with the teachers. Those pupils without proper breakfasts were more often fatigued and inattentive than those eating better breakfasts. Blood sugar values could not be correlated. Tucker,[60] in a Kansas school survey, reported that almost 8 per cent of the children ate no breakfast. Tuttle, Wilson and Daum[61] found that work output and reaction time were often unfavorably affected by an inadequate

breakfast, especially where coffee was used as a substitute for nourishing food. An excellent study on blood sugar values in relation to various types of breakfasts has been published,[62] and the inadequacy of many meals served in industrial plants has been carefully documented by Goodhart.[63]

Supper. In addition to scanty breakfasts many individuals eat a hasty and ill-planned lunch and then try to make up the deficiency by gorging themselves with a heavy dinner at the supper hour. This is most unnatural. After a night of serene rest the digestive organs are best able to receive a heavy meal; therefore, breakfast should be substantial in quantity and quality, to prepare the body for the energy and maintenance needs of the day. At noon, when half of the daily work is completed, the body is still able to handle a sizeable meal and requires further energy for the remaining duties of the day. By evening, after a day of frustrations in modern life and when the body is least able to handle a heavy meal, most Americans eat their heartiest meal. If the individual retires at a reasonable hour the food is still in the stomach, being only half digested, and the would-be sleeper may wonder why he has insomnia and other distresses. We recognize that much of our modern industrial life seems almost to necessitate a heavy evening meal; nevertheless, where possible and where necessary, our patients will be benefited nutritionally if some adjustment is made in this matter. In the present series we found that 63 per cent of the patients ate their heartiest meal in the evening.

Snacks between meals. Since gastric digestion requires four to five hours, at least five hours should elapse between each meal. Digestion is essentially a series of chemical processes and it is common knowledge that imperfect results are obtained in any chemical reaction if ingredients are added now and then to suit a fickle fancy. Any housewife knows that a poor plate of beans will result if intermittently a spoonful of dry beans is tossed into the kettle after the main mass is half cooked. Yet, that is the way many persons treat their digestive organs and processes. To make matters worse the foods most commonly eaten between meals are the sweet and devi-

talized foods. In the present series 48 per cent of the individuals ate nutrients between meals. Sometimes just the correction of this one habit will result in much improvement in health.

Laxatives. Roberts[16] has stressed the seriousness of the laxative habit. Not only will mineral oil dissolve out fat soluble vitamins and cause them to pass out in the feces, but as much as 35 to 60 per cent of the oil may be absorbed into the bowel wall, blood stream, lymph nodes and even the liver.[64] Other laxatives likewise interfere with normal bowel motility, digestion and absorption. The laxative habit is usually easy to relieve by assuring a normal diet with adequate fruit content, and plenty of water taken one-half hour before meals. Even an otorhinolaryngologist who corrects his patients' living habits and habits of eating may have gratified patients who are no longer enslaved to the laxative habit or the enema tube. In our series 53 per cent used laxatives.

Drugs. A relatively small percentage of our series engaged in self-medication, other than laxatives, on a large scale, so we did not tabulate them; nevertheless, it is an important point in human nutrition. Whether self-administered or taken upon a physician's prescription, such drugs as the sulfonamides can reduce the vitamin C content of body tissues (see report by Pelner[65]). Even penicillin has been regarded as an antivitamin in its chemical degradation process.[66] The effect of antibiotics in destroying nonpathogenic bowel bacteria that synthesize certain vitamins is of course so well known as to require no further comment. The relationship of acetyl-salicylic acid to vitamin K has been discussed earlier in this paper.

Special diets. In our history we inquire into the use of so-called "special diets" in the past. Many patients are placed on such diets (often printed stock diets for certain diagnoses) by physicians or dietitians and then adhere to them indefinitely, long beyond a reasonably intended time. Spies[7] has shown by analyzing various "special diets" commonly used by physicians that essentially all of them are deficient diets. The obvious conclusion must be that if such diets are used for any

length of time, instead of *curing* disease, they will produce disease. There is no "special diet" that is a good substitute for a nutritious diet.

The eyes. Earlier in this presentation the importance of vitamin A in corneal integrity and in visual dark adaptation has been discussed. Our series finds that 63 per cent of the patients complained of visual distresses (see page 3 of history). Interestingly enough, there were a number of instances of very obvious and severe dietary inadequacy in which the patients had no visual complaints. It must also be kept in mind that refractive errors, industrial lighting situations and many other factors can produce eye distress. Such distress is not necessarily nutritional in origin, but in occasional cases may prove suggestive.

Symptoms referable to the nervous system. In our history (see page 4) we elicit evidences of nervous system disturbances such as a feeling of nervousness, tension, worry, mental sluggishness, etc. While these are nonspecific complaints and may indicate a functional disturbance or possibly even a reasonably normal reaction to the great frustrations and perplexities of present-day life, they still may be suggestive of nutritional failure. An excellent review of the available literature on the subject was recently presented.[67] The review points out that it has been shown by a number of workers that symptoms of psychoneurosis can readily be produced in human experimental avitaminosis B, particularly with reference to thiamin deficiency. The nervous system findings in pellagra are well known. The depleting effect of refined carbohydrate diets on the B vitamins has previously been discussed.

Jolliffe[68] in an excellent paper has recognized the problem and has outlined a program of vitamin therapy on a broad nutritional base to be used in neuropsychiatric disorders. In my series 79 per cent of the patients recorded symptoms referable to their neuropsychiatric life. I do not claim that these symptoms prove the presence of nutritional failure, but they are symptoms which should cause us to search for evidences of nutritional inadequacy.

SUMMARY.

1. Nutritive failure in varying degrees is prevalent in possibly half or more of the American population. Most deficiencies are multiple and involve more than one nutrient element.

2. Many of these individuals will present themselves as patients to otorhinolaryngologists because so many of the symptoms of malnutrition evidence themselves in this field of practice.

3. There is a growing awareness of this problem among otolaryngologists.

4. A careful nutritional study should include a careful nutritional history as well as physical examination and proper laboratory tests.

5. A rather simple history form which conserves the physician's time has been presented.

6. In a survey of 500 such histories the excessive use of refined carbohydrates and gross irregularities in mealtime habits were found to be among the more outstanding dietary errors.

7. Nutritional guidance can be made simple, yet effective, in clinical practice.

CONCLUSIONS.

Modern otorhinolaryngology requires that nutritional investigation and guidance be made available by the physician to the patient, to the end that optimum health may be regained and also maintained.[7]

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CARCINOMA IN SITU IN OTOLARYNGOLOGY.*†

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Carcinoma *in situ*, or preinvasive carcinoma, is being diagnosed with increasing frequency in many fields of medicine.[32] This is a logical development of the emphasis being placed upon early diagnosis as one of the fundamentals in the concerted campaign against cancer. These early carcinomas were first detected in the skin and in the uterine cervix.[1,81] Comparatively recently carcinoma *in situ* is being found in other areas, including the field of otolaryngology. Several groups of significant numbers are being collected in otolaryngological centers;[1] however, at this time and until the concept of carcinoma *in situ* has been more widely attained, whenever such a diagnois is made by the pathologist, immediately there may be injected into the clinical problem a doubt as to the exact meaning of this pathologic terminology.

A precise understanding of the significance of carcinoma *in situ* is a prerequisite to selection of proper treatment. How does it differ from conventional carcinoma? Is it actually malignant? Will it invade, and how soon? Should it be treated differently from other types of carcinoma? These are some of the questions which complicate the choice of therapy. The purpose of this paper is to review some of the basic steps in the evolution of this diagnosis, in so doing to clarify the concept of this term, and by selected case reports to illustrate some of the problems in the management of carcinoma *in situ* in otolaryngology.

Extensive literature has accumulated on the subject of cellular studies and preinvasive carcinoma. Increasing knowl-

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edge of the characteristics of the malignant cell, accidental discovery of unsuspected carcinoma *in situ*, especially in the cervix, and cytological studies stimulated by Papanicolaou have made possible the rapidly increasing number of cases of carcinoma *in situ* which are being diagnosed in practically all fields of medicine.

The clinical importance of recognizing early cancer is obvious and has been stressed by numerous authors. Mallory has said that carcinoma *in situ* should be completely curable as it is only after invasion occurs into lymphatics and blood vessels that metastases become a probability.[52] As long ago as 1921 Cullen reported an unsuspected case of carcinoma of the cervix which was discovered accidentally while the body of the uterus was being curetted for hemorrhage caused by hyperplasia of the endometrium. He stated that "It is to the early diagnosis of carcinoma of the cervix that we must look for future success."[16] Meigs says that the early diagnosis and treatment is a prerequisite for cure, and emphasized that delay is responsible for a majority of deaths from cervical cancer.[57] Younge and associates mention the first carcinoma of a series which was discovered by biopsy only after the cervix failed to heal following cauterization. This case of carcinoma *in situ* was seen to progress to invasive carcinoma in 11 months in a deliberate experiment.[90] Fremont-Smith, Graham and Meigs express the opinion that until the cause or causes of carcinoma are discovered, the only way to control cancer is by early diagnosis and eradication at its inception.[31] Foote and Stewart state that it is impossible to detect carcinoma *in situ* with the naked eye even though the pathologist knows the specimen contains a cancer.[29] McCollum writes that diagnosis in the preinvasive stage is necessarily microscopic because most of the gross changes depend upon invasion.[56] Smith maintains that the diagnosis of preinvasive cancer depends upon the ability of the pathologist to discern when cells are assuming or have developed malignant characteristics.[81] An early diagnosis, therefore, requires a complete knowledge of the microscopic characteristics of malignant cells.

The most extensive studies have been on the cervix;[1] however, squamous carcinoma *in situ* has been reported from skin,[7,8] oral cavity,[9] larynx,[11,14,33,76] esophagus,[43] bronchus,[6,22,23,70] kidney,[25] vulva,[44,45] and adenocarcinoma *in situ* has been recorded from stomach,[52] large bowel,[6] appendix,[56] breast[28] and endometrium.[39] A brief resumé of selected studies will simplify the problems of understanding and treating carcinoma *in situ*.

Beginning approximately a hundred years ago, sporadic and confused attempts were made to identify the characteristics of neoplastic cells.[84] Hauptmann dates studies of the cancer cell from the advent of Virchow's cellular pathology studies.[36] Zemansky relates that Quincke, in 1875, published careful histological observations on chylous ascitic fluids, and in 1882 reported examinations of stained smears of a large group of peritoneal and pleural fluids in inflammatory and neoplastic conditions. In 1895, Reider reported seeing mitoses for the first time in stained smears of cells free in fluid media.[91] Although as early as 1901 Mandlebaum studied actinomycotic pus by fixation and section, it was not until 1917 that his method of cellular studies by stained paraffin sections of fixed sediment of centrifuged body fluids was published.[53]

By this time numerous other pathologists more or less independently had arrived at a similar technique.[27] More rapid methods of studying cells were coming into use also about this time. In 1910 and again in 1923, Shaw published a rapid method of staining frozen sections of fresh tissues in the operating room. Frozen by ether or ethyl chloride, tissue was cut and stained by dilute Loeffler's methylene blue. He had performed 540 examinations up to the time of the latter publication.[79] Closely following this, Dudgeon and Patrick reported 191 correct diagnoses out of 200 studies by a wet-film method of staining fixed scrapings from cut surfaces of tumors. Dudgeon and Barrett, in 1934, wrote that over 1,000 cases had been studied by this wet-film method. A year later Dudgeon and Wrigley reported their demonstration of malignant cells in sputum in 68 per cent of 38 cases of proven carcinoma of the lung by this same wet-film method.[18-20]

In this country, in 1912, Bowen described carefully two cases of atypical epithelial proliferation which he considered to be precancerous dermatosis. By 1915 he reported the sixth such case.[7,8] After much criticism and skepticism these cases of Bowen have finally been recognized as intraepidermic carcinoma or preinvasive carcinoma, and, therefore, were apparently the first cases of carcinoma *in situ* to be described.[86]

McCarty has written repeatedly on studies of the differential characteristics of the cancer cell. He points out that the nucleoli of cancer cells are larger in proportion to the nucleus than in nonmalignant cells. These observations have been consistently found by his associates using a method of accurate cell measurements.[47-50]

Thirty-five years ago Papanicolaou, of the Department of Anatomy, Cornell Medical College, began the study of morphologic and cyclic variations of cells during the sex cycle in guinea pigs by means of vaginal smears.[60] The usefulness of this method of study was immediately evident. In 1923, he began a systematic study of the human sex cycle by daily vaginal smears.[61] He noted striking abnormalities of cancer cells and their nuclei. He was immediately aware of the potentialities for diagnosis in the human. Although his cytological technique was successful in diagnosing a case of uterine carcinoma which he reported in 1928, clinical men failed to recognize the possibilities of this method. Papanicolaou lost enthusiasm and returned to his former work for a time.[66,68]

Cullen, in 1921, reported an unsuspected early cancer of the cervix discovered by pathologic study of a specimen removed at operation.[16] This has been considered one of the earliest reported cases of carcinoma *in situ*.[90] This term was unknown at that time, and was not used by Broders until 1932.[52,53] Pemberton and Smith had begun to talk of noninvasive carcinoma about 1929, but their ideas were not acceptable to pathologists in general at that time.[51]

The introduction of aspiration or needle biopsy is another attempt to recognize cancer at an earlier stage on the basis

of the characteristics of malignant cells. Martin and Ellis describe the Mixter punch, a blunt tipped trochar used for biopsy of brain tissue in the early years of this century. Ward suggested needle puncture and aspiration of lymph nodes in 1912. Goeller used a special cutting-tipped trochar in 1920 for prostatic biopsy, which Martin and Ellis considered more traumatic than an 18 gauge needle. Guthrie aspirated Hodgkin's nodes in 1921. Martin and Ellis began to use a sharp 18 gauge needle in 1926 for aspiration biopsies. The aspirated material or plug of tissue was smeared, fixed and stained. They reported 65 cases, almost half of which were from cervical lymph nodes. They describe two cases with fixed, indurated masses anteriorly and low in the neck associated with dyspnea, where the differential diagnosis of laryngeal from thyroid carcinoma was made on finding squamous cells by aspiration biopsies of the cervical masses.[54] In 1927, Forkner described the use of a dental brooch introduced through an 18 gauge needle for the purpose of securing material from lymph nodes for supravital staining studies.[30] Ferguson reported needle puncture and aspiration biopsies of prostatic neoplasms in 1930.[24] In 1931, Sharp was diagnosing primary carcinoma of the lung by needle aspiration through the chest wall in neoplasms inaccessible to the bronchoscope,[78] and in the same year Coley, Sharp and Ellis related their successful diagnosis of bone tumors by the aspiration method.[15] In 1933, Stewart reported approximately 2,500 cases of aspiration diagnosis of tumors without unfavorable result.[82] Also during these years pathologic studies, especially by sections of the surgically removed uterine cervix, were helping to clarify the ideas of preinvasive cancer cells.

It is interesting and significant that many authors refer in recent years to Bowen's Precancerous Dermatosis, first described in 1912. Sutton and Sutton wrote that Bowen's Disease or Intraepidermal Carcinoma of Bowen's type has been widely recognized in Europe since 1922, and that Wise, in 1929, found that this was a definite, distinct entity with histologic features peculiar to itself and indicative of intraepidermal carcinoma. The Suttons were convinced that Bowen's

disease is true carcinoma, although it remains superficial for a long time.[33] James Ewing describes it as "a peculiar, very chronic lesion of the epidermis, which doubtless represents a specific form of precancerous keratosis," occurring usually in elderly people, being single or conglomerate, localized or widely distributed, dull red, scaling or crusting, painful papules or elevated nodules in the skin, which may progress slowly, requiring 10 to 30 years before becoming malignant.[21] Mallory says that the "evolution into invasive cancer seems to be constant in Bowen's disease. The irreversible character of this evolution, and the constant presence of certain histologic features common to malignant neoplasms but foreign to inflammatory reactions gradually aroused the suspicion that such conditions represented not merely potential foci of carcinogenesis but actual carcinoma in a preinvasive stage."[52] He states that this theory was crystallized in 1933 by Schiller, who found a case of preinvasive carcinoma of the cervix while seeking a means of diagnosing carcinoma early by studying microscopic sections of all cervices removed at the Gynecological Clinic at the University of Vienna. One of his cases presented an early unsuspected noninvasive carcinoma within the cervical canal, hysterectomy having been done because of an adnexal tumor. He called this "young carcinoma," or the "early stage of carcinoma." He wrote that their experience "demonstrated that the smallest carcinomatous layer without downgrowth will later, if allowed to grow, develop into deeply penetrating carcinoma" and eventually will ulcerate superficially. He compared his cases with Bowen's to whom he credited the discovery of such findings in relation to skin.[73] Five years later Schiller again compared the biological and morphological similarity between Bowen's disease and carcinoma of the cervix. He stated that there may be many months before any appreciable change in the size of the lesion can be detected. "Rapid and excessive growth which has always been ascribed to malignant tumors, apparently does not occur until the carcinoma has reached a definite size, a certain amount of superficial disintegration, and some invasive growth."[74] Mallory writes that Broders independently arrived at the same concept and coined the phrase "carcinoma

in situ," "to describe the expressions of faith by pathologists that a given segment of epithelium has undergone the initial stage of an irreversible sequence which can end only in frank invasive carcinoma." He believed it was impossible to foretell when invasion would begin.[52] Bowen's first case did not invade until after a period of 19 years, yet Schiller's case required only seven years before invasion. Willis, writing of further studies on the origin of carcinoma of skin, also refers to Bowen's disease, and states that the so-called precancerous dermatosis is now considered to be genuine carcinoma *in situ.*[86] Brighton and Altmann speak of superficial squamous epithelioma of Bowenoid type in reporting cases of intraepithelial spreading carcinoma of the upper respiratory and alimentary tracts.[9] In recent years the many favorable references to Bowen's work can only signify recognition of the importance of his early contribution to the study of preinvasive cancer.

In 1932 Broders emphasized that above everything else the microscopist must consider the character of the epithelial cells in order to arrive at a correct diagnosis. He said that the day is past when epithelium can be considered nonmalignant because it has not penetrated the basement membrane, or malignant just because it is not confined within the basement membrane. In the latter sense he referred to benign penetrating epithelium in benign granulomatous ulcers such as in tuberculosis, syphilis and actinomycosis. He said, "The diagnosis of carcinoma *in situ,* as of any carcinoma in general, is based chiefly on altered cellular characteristics in contradistinction to cellular situation." He described the nuclei of malignant cells, in contrast to those of normal cells, as having greater avidity for basophilic stains, as usually increased in size both actually and as related to the cytoplasm, as frequently irregular, not infrequently in a state of pathologic or atypical mitosis, and occasionally observed in groups in the form of tumor giant cells. He believed carcinoma *in situ* may assume any type, squamous, basal or adenocarcinoma; it may occur in any location in which carcinoma arises, and may be of any degree of malignancy. He stated that in a biopsy of

carcinoma with infiltration, the diagnosis will be easy; but if carcinoma *in situ* alone appears, it is necessary to recognize the malignant cell characteristics because the failure to do so is an error of omission fraught with grave danger to the patient.[10]

Great credit must be given to Papanicolaou for his studies of cytology. He writes that the idea of staining exfoliated cells was not new because Pouchet studied vaginal cells a hundred years earlier and Hampelon stained sputum 60 years ago. As we have related, Papanicolaou became discouraged at his inability to interest clinicians in his cytological method of cancer diagnosis by vaginal smears, and returned to his previous work; however, in 1937, he became associated with Dr. Herbert Traut in the study of cancer once again. Vaginal smears were made routine in practically all gynecological patients in the Women's Clinic of the New York Hospital. In a monograph in 1943, Papanicolaou and Traut reported studies of vaginal smears of 3,014 adult women. There were 127 cases of carcinoma of the cervix and 52 cases of the fundus. The cytological diagnosis by the vaginal smear technique failed in 3.2 per cent of the cervical and in 9.3 per cent of fundus cases. They expressed the hope that cytological studies of smears from the cervical canal would be more accurate. Later Papanicolaou reported that endocervical and endometrial smears reduced the chance of overlooking carcinoma of the cervix and endometrium.[65,66,68]

Since the monograph of 1943, Papanicolaou's work has been confirmed by a number of other gynecological investigators.[66] Fremont-Smith asserts that his group from Harvard was the first to corroborate Papanicolaou's cytological studies. They wrote that biopsy is the best method of diagnosis; that there is exfoliation of cells from every epithelial surface of the body into the surrounding medium; that the cytological diagnosis of malignant tumors can be made on any cancer involving the free surface of any part of the body; that Meigs has presented evidence proving cytology theoretically sound, statistically reliable and superior at least in certain respects to biopsy. Meigs showed that identical cells could be obtained by secur-

ing smears of malignant tumors at operations or from smears of body fluids of the patient bearing the tumor. The diagnostic error of one group of 220 patients having 46 proven cancers was as low as 2.2 per cent.[31] There have also been rather numerous articles describing modifications of Papanicolaou's technique, and setting forth criteria of malignant cell characteristics.[4,35,36,40,65]

The Papanicolaou technique depends upon immediate fixation of smears while still wet. Equal parts of absolute ethyl alcohol and ether is the preferred fixative. This method avoids the distortion that accompanies drying of cells.[64] Staining technique accentuates nuclear detail, while maintaining cytoplasmic transparency. Smears now are being made of any body secretion, or of centrifuged sediment of thin body fluids or washings of epithelial surfaces. Sediment adheres to albumen covered slides, and fixation is carried out before drying can occur. One advantage is technical simplicity, and Papanicolaou also points out that it is not very expensive or inconvenient to patients.[68] There have been numerous reports of modifications of this technique,[5,26,36,62,72] although Hunter and Richardson maintain that the value of Papanicolaou's stain cannot be overemphasized.[40]

Papanicolaou affirmed that the diagnosis of cancer by cytological studies should be "based on strong, substantiative evidence offered by sufficient pathognomonic cells" and not merely on a few cells. He maintains that negative or inconclusive tests should be repeated. Among the advantages that he has described are: *1.* its simplicity and inexpensiveness; *2.* its reliability in hands of experienced cytologists; *3.* that it permits early recognition of hidden or incipient neoplasms, such as carcinoma *in situ* by exfoliation before the onset of ulceration; *4.* its suitability for a screening method on large numbers of patients; *5.* that it is a valuable complement to biopsy or curettage because the cytological method covers a larger area; *6.* it may reveal carcinoma although biopsy may have failed; and *7.* it has unique value in following operative and radiologic treatment. On the other hand, this method does not indicate the grade of malignancy, does not supply infor-

mation about metastatic activity or relationship to adjoining normal tissue, and the average time required for carefully studying a smear is longer than for a pathologic section. The identification of carcinoma by the cytological method depends upon recognizing the characteristics of the malignant cells which are constantly being exfoliated from malignant neoplasms having a free surface.[68]

The morphological character of malignant, yet not invasive, epithelium is characteristic and is so consistent that not infrequently a brief low power examination is all that is necessary to recognize its neoplastic nature. Younge asserts that preinvasive carcinoma resembles the type of epithelium frequently found at the edge of an invading cancer.[89] From his cytological studies Papanicolaou says that cancerous cells should show: *1.* Anisocytosis; *2.* Anisokaryosis; *3.* Hyperchromasia (abnormally densely stained karyosomes and nuclear membrane); and *4.* abnormal mitotic figures.[64] Reagan emphasizes that alterations in the appearance of the nuclear chromatin are the most important morphological feature in identification of the tumor cells, and that nuclear characteristics are important in differentiating cells derived from carcinoma *in situ.* These nuclei are usually only slightly larger than the nuclei of the normal parabasal type cells of comparable size.[72] Papanicolaou further describes his criteria for recognizing cancer cells as both cytological and histological. This includes: *1.* variation in size of nuclei many of which are enlarged, both absolutely and relatively to the amount of cytoplasm; and *2.* variation in shape of nuclei which also figure prominently in identification of tumor cells. He includes other nuclear characteristics as abnormal clumping of chromatin, hyperchromasia, and enlargement of nucleoli. Less reliable are cytoplasmic criteria, but frequently there will be vacuolization and at times cytoplasm will show engulfment of other cells.[75] In spite of the apparent simplicity of recognizing cancer cells, there are numerous warnings that an experienced cytologist is absolutely necessary in this method of diagnosing carcinoma.[31,63,72,84]

After the cytological technique in the diagnosis of cancer of the female genital tract became well established, Papanicolaou says it was logical to attempt the same method in study of other systems.[66,68,70] This has been used successfully in study of centrifuged sediment of prostatic secretions, urine, sputum, bronchial smears and washings, gastric content, gastric-duodenal content and esophageal washings, laryngeal smears, nasopharyngeal smears, rectal mucus, acitic and other fluids. There are by now numerous reports of carcinoma *in situ* discovered on the basis of cytologic smears in various organs.[2,3,17,22,23,37,41,42,43,46,67,69,70,75,87,88]

There existed a reticence on the part of some pathologists to diagnose carcinoma without invasion.[28,36,59] Foote and Stewart comment, "This is hardly logical since it is so much more reasonable to conclude that if the tumor had not been a carcinoma prior to infiltration, then infiltration would not have occurred."[28] Moreover, various histogenetic studies link intraepithelial carcinoma with invasive carcinoma. This is undoubtedly true in Bowen's disease and in carcinoma of the cervix.[52,73,74,81] Younge, commenting on Smith and Pemberton's cases reported in 1934, points out that five of the 16 cases were not diagnosed carcinoma *in situ* on the original biopsy, but only on reviewing the original sections three and one-half to twelve and one-half years later when these patients began to develop invading carcinoma of the cervix. Younge describes two additional cases in which the original biopsies were considered benign, and reclassified only two to three years later when invading carcinoma developed.[89] Pund and associates studied 1,200 surgically removed cervices. They concluded that the preinvasive stage becomes invasive after a variable latency averaging five to six years, which seems to be the length of time required for endocervical carcinoma to develop sufficient penetration and ulceration of the vaginal surface of the cervix to become obvious.[71] Younge, Hertig and Armstrong describe an early case of carcinoma *in situ*, diagnosed by biopsy because healing would not follow cauterization of the cervix, and in which the carcinoma became invasive in 11 months.[90] Hertig, Sommers and Bengloff

describe repeated biopsies on 11 patients showing sequential endometrial changes which advanced from carcinoma *in situ* to typical carcinoma.[39] Black and Ackerman believe that qualified, independent investigators,who have described about 30 cases, have well documented evidence that carcinoma *in situ* of the cervix has been followed by invasive carcinoma.[6] Black and Ackerman have studied epidermoid carcinoma *in situ* of the bronchi and lungs and concluded that "evidence has accumulated that epidermoid and undifferentiated tumors of the major bronchi may arise first as islands of carcinoma *in situ*." They describe the initial phase as a progressive metaplasia of the surface epithelium with cellular hyperactivity in the basal layer; increased mitotic activity of the basal layer produces packing of nuclei, increased stratification, and thickening of the epithelium; mitotic activity increase as the basal cell activity extends closer to the surface. Finally all layers are involved, and the ciliated superficial layer no longer is present. Preinvasive carcinoma of the bronchus is present with the changes extending through all layers to the surface from the intact basement membrane. They reviewed 60 recent pneumonectomy cases and found that in 35 per cent of these either marked anaplasia of intact epithelium or frank carcinoma *in situ* was closely adjacent to invasive carcinoma.[6] There seems little reason to doubt that preinvasive carcinoma, if uninterrupted by effective treatment, will eventually cause the death of the host.[32]

The laryngeal epithelium may undergo the same type of progressive cellular alteration as occurs in the uterine cervix with the development of increasing activity in the basal layer, increased mitotic activity, packing of nuclei, increased stratification, and development of carcinoma *in situ*;[1] therefore, it should be possible to discover carcinoma of the larynx in the preinvasive stage when the best chance for cure exists.

Such reports are beginning to appear in the otolaryngologic literature. Clerf reported an unusual case of carcinoma of the larynx diagnosed by Mackenty in 1927. Laryngectomy was refused until 1938 when it was performed by Clerf. After review of the tissue and original biopsy by another path-

ologist, it was thought that this might have been a case of "Bowen's disease of the larynx."[14] Brighton and Altmann reported six cases of carcinoma *in situ* from the upper respiratory and alimentary tracts. There were two cases from the larynx and one each from nasal septal mucosa, tonsil, oral mucosa and pharyngeal mucosa.[9] Snitman advocated serial section of all removed laryngofissure masses to avoid incomplete removal of tumor, and to find previously unrecognized tumor. He reports a case with carcinoma completely removed from the anterior and middle thirds of the left cord, but with carcinoma *in situ* in the anterior portion of the right cord.[76] Gordon found carcinoma *in situ* underlying keratosis of the larynx.[33] Brunner has reported a case of superficial epidermoid carcinoma of the larynx associated with trichinosis of laryngeal muscles.[11] For several years Papanicolaou's technique has been employed by Siebert for studying laryngeal and nasopharyngeal smears.[80] Bryan and Bryan, who at first utilized Papanicolaou stains to study epithelial cells during upper respiratory infections,[12] since have studied many smears from various areas of the upper alimentary and respiratory tracts.[13] Their reports have been of great help in diagnosing malignant neoplasms, and in evaluating the results of irradiation treatment. Schoolman advocates both biopsy and cytologic smears from the nasopharynx in order that the value of the Papanicolaou technique in this area may be determined.[77] Such developments indicate that earlier recognition of otolaryngological malignancies may be expected. In hope of stimulating interest in problems associated with carcinoma *in situ* in otolaryngology, the following cases are described:

CASE REPORTS.

Case 1: L. M. M., a 27-year-old, single, white female laboratory technician, a private patient, complained of slight hoarseness for about six months when first seen in October, 1948. She was worried about cancer. She had a quiescent, compensated rheumatic heart and normal blood pressure. Physical examination otherwise was entirely normal except for the presence of two very small, smooth laryngeal nodules, one on the free edge of each vocal cord at approximately the junction of the anterior and middle thirds. These nodules were in contact on phonation. The color of the cords was a normal pearly white and that of the nodules was a flat light gray. There was no redness surrounding the nodules.

She entered McMillan Hospital Oct. 19, 1948. The nodules were removed the following morning without difficulty by the suspension method, using laryngeal cup forceps. The nodule on the left cord was minute, and the larger nodule on the right cord was about the size of the head of a common pin. There was no clinical suggestion of malignancy. Because of the possibility of papilloma, the denuded areas of mucous membrane were carefully touched with a very small amount of 25 per cent silver nitrate solution on a laryngeal applicator.

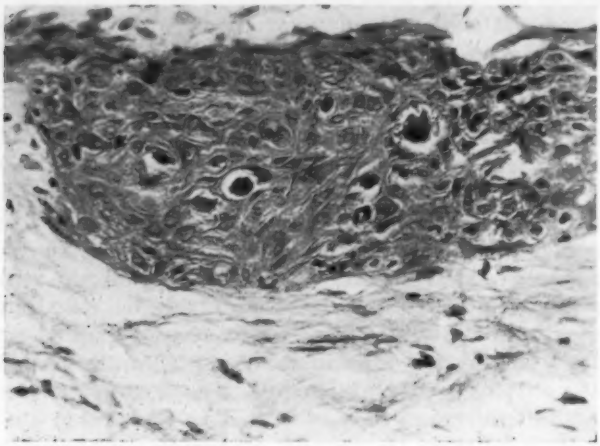

Fig. 1. Case 1, L. M. M. Carcinoma in situ of left vocal cord, Oct. 20, 1948. 300×.

We were concerned, indeed, the following day to receive a diagnosis of carcinoma *in situ* of both cords by Dr. Parker A. Beamer, of the Washington University Pathology Department. This diagnosis was confirmed also by Dr. Lauren Ackerman and Dr. Robert A. Moore.

The specimens consisted of three pieces from the left cord, the largest of which measured 2 x 1.5 x 0.5 mm., and two pieces from the right, the larger of which was 3 x 2 x 2 mm. The squamous epithelium bilaterally showed disarrangement of its structure. There were large cells, many of which showed mitotic figures and tended to arrange themselves perpendicularly to the surface. There was no invasion below the basement membrane. There seemed to be no doubt that carcinoma *in situ* was bilateral.

She was presented at the weekly conference of the Department of Otolaryngology, Washington University, for therapeutic recommendations. Diathermy excision under suspension laryngoscopy as advocated by Dr. Gordon New was considered. Dr. Ackerman felt that a curative dose of X-radiation could be recommended on the basis of the pathologic cell changes; however, because of her hospital association and availability for daily laryngeal study, it was decided to keep her under the closest

observation, to rebiopsy if any suggestion of a recurrence could be seen, and to defer any specific treatment at that time. Her larynx healed nicely. Daily laryngeal examinations gradually became weekly, then every two weeks, then monthly. Her voice is good. There has never been any suggestion of recurrence. On one or two occasions she has been slightly hoarse for a few days during a head cold. She remains under regular observation.

This case clearly illustrates the necessity for careful pathological study of even the most minute laryngeal specimen. It also demonstrates the feasibility of close observation of the larynx for evidence of recurrence because of its accessibility. If recurrence is ever suggested, immediate cytologic and histologic studies will be indicated; if cancer cells are found, an adequate type of treatment must be instituted without delay.

Case 2: G. L. P., a 54-year-old white male, private patient, a masseur, complained of intermittent huskiness for about three years, which had been worse with colds. There was no history of weight loss, cough, or loss of appetite or strength. He was a moderate cigarette smoker. When first seen in April, 1950, he was slightly hoarse. There was moderate diffuse chronic thickening of both true cords, which were dull gray. The laryngeal appearance suggested hyperkeratosis. In addition, the left cord presented a slightly yellow area, 3 to 5 mm. in diameter, on its superior surface near the vocal process, and a small gray nodule on the free edge near the junction of the anterior and middle thirds. The epithelium of the right true cord was similarly and uniformly thickened. General physical condition was good. Blood pressure was 120/70. A chest film was negative.

He was admitted to McMillan Hospital. The following day, April 15, 1950, suspension laryngoscopy was uneventful. The thickened membrane stripped easily from the right cord, and was removed in four pieces from the left cord with laryngeal cup forceps. There was minimal postoperative discomfort.

The pathological diagnosis was hyperplasia on the right and carcinoma *in situ* on the left. Two pieces from the left cord showed thickened but otherwise normal epithelium with edema of the stroma. The other two pieces were described by Dr. Edward B. Smith, of the Washington University, Department of Pathology, as showing "very interesting epithelium with thick parakeratotic corneum and acanthotic underlying prickle cell zone. The latter zone forms wide, rounded rete pegs in which two abortive epithelial pearls and several mitotic figures are found. One mitotic figure is atypical. The basal layer is intact in every sector."

His larynx was watched carefully during healing. The cords regained a fairly normal appearance and his voice was good. Approximately four months later he developed a fever and cough. This apparently was due to a second malignancy developing very rapidly in the right hilar region. He would not consent to endoscopic examination until late in October, 1950. At bronchoscopy the tumor was not accessible and the vocal cords remained entirely normal in appearance. Exploratory thoracotomy elsewhere revealed adenocarcinoma.

This case history illustrates the almost simultaneous development of distinctly different, primary malignant tumors in the same patient, the laryngeal being of squamous cell origin and the bronchogenic tumor showing adenocarcinoma. This man did not survive sufficiently long after the removal of his laryngeal carcinoma *in situ* for any conclusions to be

drawn about the tendency of the laryngeal tumor to recur. This patient is one of many in various fields of medicine who have been shown in recent years to have developed distinctly different and secondary primary malignancies.

Case 3: M. S. B., a 58-year-old white male, a grocery clerk, was admitted to the Washington University Otolaryngology Clinic with a 15-year history of hoarseness, which had been worse for three or four years.

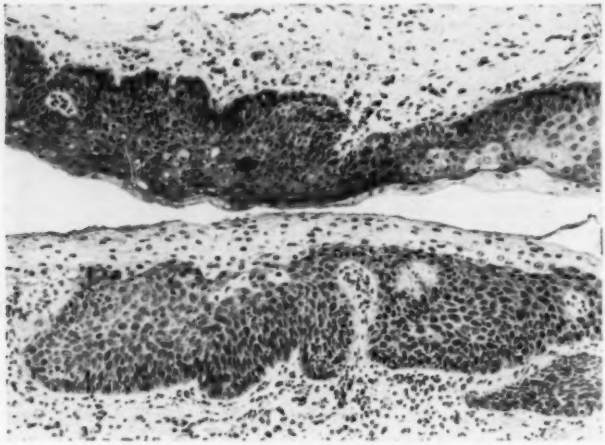

Fig. 2. Case 3, M. S. B. Sections from laryngofissure block, Dec. 19, 1950. Epithelial layer above, showing changes throughout its entire depth; adjacent epithelial layer below, demonstrating marked basal layer changes approaching the surface; basement membrane remains intact.

There was a small laryngeal nodule projecting medially from the middle third of the right true cord. This was smooth. There was no ulceration. The nodule was in contact with the left cord on phonation. His blood pressure was 210/110 and there was moderate cardiac enlargement.

Oct. 25, 1950, the laryngeal nodule was removed at McMillan Hospital and showed epidermoid carcinoma *in situ*. Rebiopsy of the right cord was done Dec. 5, 1950, and the report again was carcinoma *in situ*. Anaplasia and hyperchromasia in the epithelium was confined within the basement membrane. There were a few broad papillae extending into the stroma. On Dec. 19, 1950, a right laryngofissure was done. Noninvasive carcinoma was present in several areas of the surgical specimen. There has been no apparent recurrence to this time.

This case is included in this report through the courtesy of Dr. J. Ogura, Dr. J. West and Dr. L. Gurnee, and illustrates the occurrence of carcinoma *in situ* of a laryngeal nodule and of that side of the larynx in a patient whose hoarseness had existed for at least 15 years. This preinvasive cancer was eradicated by adequate treatment.

Case 4: A. M., 78-year-old white male, proprietor of a barber shop, private patient of Dr. M. F. Arbuckle, through whose courtesy this case is described, was admitted to McMillan Hospital June 2, 1948, for laryngeal biopsy because of fusiform red thickening of the right true cord. Hoarseness was first noted seven months earlier after he had strained his voice in public speaking. Hoarseness had not been constant but was noted if he talked for any length of time. General physical condition was good. Blood pressure was 150/90. He had suffered some previous anginal symptoms. Electrocardiogram was normal. Several biopsy specimens were taken through the anterior commissure laryngoscope. The clinical appearance was not very suggestive of malignancy. Three pieces of tissue were described as soft, pinkish or grayish. The largest specimen was 4 x 3 x 2 mm. The pathological description included fibrous tissue linked by respiratory and metaplastic epithelium, an intact basement

Fig. 3. Case 4, A. M. Original biopsy of right true cord, June 3, 1948. Epithelium was described as metaplastic. Diagnosis of carcinoma in situ was made on review by another pathologist 15 months later, after frank invasive carcinoma was almost beyond the limit for laryngofissure. Failure to consider original biopsy tissue malignant was an "error of omission fraught with danger" to this patient. 85×.

membrane, areas of hemorrhage in the fibrous tissue, and large, deeply pigmented cells. Nothing in the report suggested that the sections had presented any special diagnostic pathologic problem. The diagnosis was "laryngeal nodule."

Hoarseness continued. In September, 1949, he again consulted Dr. Arbuckle, saying that for the last five or six weeks his voice had been worse and that talking was painful at times. The right true cord was red, thickened, with an elevated, definite malignant appearing lesion. His general physical condition was relatively good. He could walk seven or eight blocks without discomfort. Blood pressure was 166/88. Dr. Edward B. Smith and Dr. Lauren Ackerman, who had not seen the original slides, were asked to review these sections. It was their opinion that carcinoma *in situ* was present in the first biopsy 15 months earlier.

Multiple biopsies were taken by Dr. Arbuckle from the right true cord on Sept. 21, 1949. The lesion extended from the anterior commissure almost to the vocal process. The cord was not fixed; the other cord appeared normal. Dr. Smith described this tissue as having a basement membrane not always intact; epithelial cells migrating into the stroma in an irregular manner; detached cords, sheets and strands deep in the stroma, showing in some places small epithelial pearly formations. The cells in general showed irregular and hyperchromatic nuclei with intercellular bridging. The diagnosis was epidermoid carcinoma, grade II.

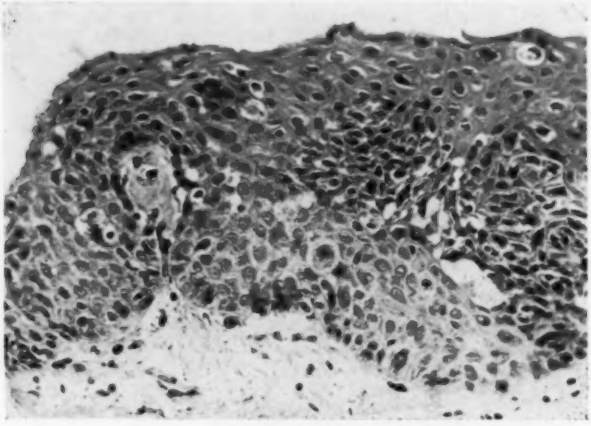

Fig. 4. Case 4, A. M. Biopsy of right true cord. Sept. 21, 1949, showing invasive carcinoma. Review of original biopsy proves that preinvasive carcinoma, or carcinoma in situ, existed in June, 1948. Preinvasive carcinoma has progressed in this larynx to frankly invasive cancer in 15 months. 255×.

Laryngofissure was performed on Sept. 26, 1949. Three or 4 mm. of the opposite cord were removed with the right laryngofissure block because of possible extension across the anterior commissure. Pathologic study showed a 3 mm. margin anteriorly. Laterally the tumor had deeply invading pegs of moderately large cells extending to within a few millimeters of the periochondrial boundary. There was a moderate number of mitotic figures. He recovered satisfactorily and is still free from laryngeal symptoms or signs of recurrence.

This case illustrates the error of omission in failing to diagnose carcinoma *in situ* on original biopsy — an "error fraught with danger to the patient," as pointed out by Broders in 1932. It also proves that preinvasive carcinoma of the larynx became frankly invasive, and was, therefore, cancer 15 months earlier at the time of original biopsy.

Case 5: L. H., 38-year-old white male, a farmer, was admitted to the Veterans Administration Hospital, Jefferson Barracks, Mo., on Oct. 30, 1950, because of backache which had been present since his wartime army service. He had thoracic kyphosis and scoliosis, and walked with

a "poker back." Also there were palpable, discrete nodes in the right side of the neck. He was seen in consultation by the otolaryngology service. Search for a cause of the adenopathy revealed a small nodular, white mass of tissue in the right glossoepiglottic fold. Tissue was removed from this area for microscopic diagnosis on Nov. 4, 1950. Sections showed typical carcinoma *in situ;* however, additional tissue was requested by Dr. Franz Leidler and Dr. Lauren Ackerman, pathologists, both of whom suggested that carcinoma may be originating in mulitple

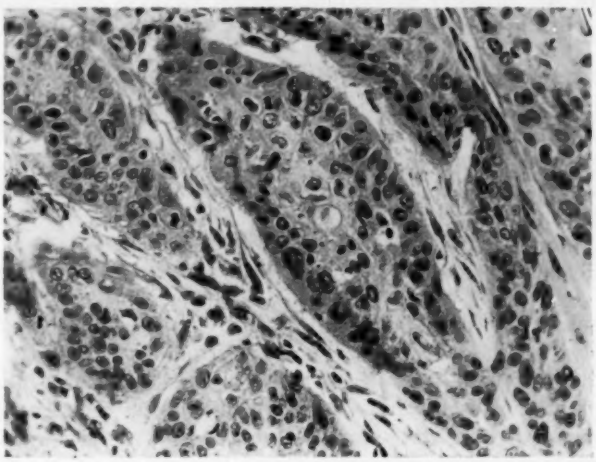

Fig. 5. Case 4, A. M. Tissue removed at laryngofissure, Sept. 26, 1949, showing invasive epidermoid carcinoma. 255×.

foci as is common especially about the oral cavity, and that this carcinoma *in situ* probably was closely adjacent to invasive carcinoma or that it was actually the advancing edge of a frankly invasive cancer. On the second biopsy, typical invasive carcinoma was found within a centimeter of the original biopsy.

This is an illustration that the possibility of inaccurate diagnosis of preinvasive carcinoma must be avoided by great care. Consultation of clinical and laboratory physicians minimize such dangers as incomplete or improper diagnoses.

SUMMARY AND CONCLUSIONS.

Carcinoma *in situ*, intraepithelial carcinoma, or preinvasive carcinoma is the earliest form of recognizable cancer. This is the initial microscopic, irreversible morphologic change in tissues which eventually will kill the host if not controlled by

adequate therapy. Alterations in cellular structure and arrangement are unquestionably neoplastic, but the essential factor of invasion is absent. The lack of penetration and the intact basement membrane have been potent deterrents in acceptance of the idea that early cancer can be diagnosed in the absence of infiltration. The preinvasive characterization infers the invasive potentiality which changes early cancer into advanced cancer.

Diagnosis of carcinoma without invasion depends upon recognition of the characteristics of cancer cells. Histologic and cytologic studies of cancer cells have been narrated. Cases of preinvasive carcinoma progressing to invasive cancer have been recounted. Knowledge of cellular changes and their potentialities clarify the concept that carcinoma *in situ* is malignant and requires courageous, prompt treatment to eradicate cancer in the early stage.

Preinvasive carcinoma is clinically invisible. More frequent biopsies and greater use of exfoliative cytology are making carcinoma *in situ* relatively commonplace. Until a better method is available for early diagnosis, reliance must be placed on constant vigilance, unrepressed suspicion, more biopsies and cytologic studies, and the collaboration of an experienced pathologist.

This paper was reviewed in the VA and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own studies and do not necessarily reflect the opinion or policy of the VA.

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SECRETORY OTITIS MEDIA.*

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Secretory otitis media is one of the most important and often most perplexing otologic problems in office practice today, although it has not received the attention it deserves.

The purpose of this paper is to review the literature, both old and recent; to discuss and attempt to evaluate certain aspects of the condition with particular interest in etiology, pathology and treatment; and to record some of my observations on 372 patients with acute, subacute and chronic secretory otitis media seen in private practice during a five and one-half year period from January, 1946, to July, 1951, inclusive.

The term secretory otitis media, as used in this discussion, is intended to include all patients who present themselves with an accumulation of serous or mucus (nonpurulent) fluid in the middle ear and usually the mastoid cavity as well. Synonyms include otitis media with effusion, serous otitis media, catarrh of the middle ear, tympanic hydrops, tubotympanic catarrh, etc. As Hoople[1] has pointed out, none of these terms by itself is adequately descriptive of this disease from an etiologic or pathologic standpoint. "Secretory otitis media" implies a true glandular secretory activity on the part of the mucosa but this is probably minimal or even absent in most cases, as will be brought out later. The term "otitis media with effusion" implies that an inflammatory process is involved, which also does not hold true in all cases. For want of a better term, however, these two will be used synonymously in this paper.

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HISTORICAL NOTE.

Many of us tend to look upon this condition as a new entity because our own experience with it is relatively recent, but upon reviewing the older literature we may well be amazed to find that Politzer[2] described it in accurate detail in 1869 and devoted 20 pages to it in the fifth edition of his book in 1909. Little has been added to our knowledge of this condition except in the fields of chemistry and allergy, and perhaps concerning the influence of the autonomic nervous system upon capillary permeability.

In 1906, Kyle[3] also discussed the treatment of catarrh of the middle ear and approved the practice of myringotomy and inflation. Some others[4] at this time were advocating the use of such measures as medicated vapors and superheated compressed air insufflated into the Eustachian tube. Ballenger,[5] in 1908, gave a good description of the mucus type of acute otitis media, as did also Phillips[6] (1919) and Kerrison[7] (1923) in their respective textbooks.

Then followed a period during which the teachings of Politzer were either forgotten or ignored, and fluid in the middle ear received little attention. With the exception of an excellent case report in 1927 by Lawson,[8] no clinical papers on otitis media with effusion appeared in the American literature until 1940. The subject of acute catarrhal otitis media had been well covered in most of the textbooks, but little had been said concerning chronic otitis media with effusion until recent years after interest in this subject had been restimulated by Jones,[9] Hoople and Blaisdell,[10] Hantman,[11] Robison[12] and others.

There is still a great deal of confusion and uncertainty concerning the etiology and pathogenesis in many of the chronic cases. As one observes and treats these patients over a period of years, there are certain questions that arise. Among those that have occurred to me, some of which have been proposed by others, are these:

1. Why do some individuals with an obstructed Eustachian tube and retracted drum develop an accumulation of fluid in the middle ear while others do not?

2. Why do some cases become chronic with little or no evidence of pathology in the nose, sinuses, nasopharynx or Eustachian orifice?

3. Why do some continue to have fluid in the tympanum in the presence of an apparently open Eustachian tube?

4. Why do the majority of reports of large series of cases come from otolaryngologists practicing in coastal areas and river valleys; or in other words, is the incidence of secretory otitis media influenced by altitude and humidity, or either of them?

5. Does determination of the protein content of the fluid have any practical value? Can one determine the origin of the fluid by determining the protein content, and is there any correlation between protein content and cytology of the fluid?

6. Are eosinophiles commonly found in the ear fluid when the tubal obstruction is the result of allergy?

The author has not found the answers to all these questions during the course of this study but has satisfied his curiosity with regard to some of them.

PATHOGENESIS.

Sterile fluid in the middle ear apparently may occur as the result of one or both of two pathological processes.

It may occur as an effusion resulting from the action of negative pressure on the thin walled arterioles and capillaries in the mucosa of the middle ear, the vacuum of course being the result of Eustachian tube obstruction ascribable to one of many possible causes. Transudation due to a vacuum alone occurs in the purest form in aero-otitis media, which Hantman[11] refers to as a "hyperacute" secretory otitis media.

Secondly, it is said that fluid may occur as a response to low-grade inflammatory reaction in the mucosa of the Eustachian tube and tympanum, secondary to infection in the nose or throat, in which case it is an exudate, but free of bacteria.

In many cases the fluid is a result of both these influences, that is, a mixture of transudate and exudate, with varying amounts of each.

ANATOMICAL CONSIDERATIONS.

A review of the anatomy and histology of the Eustachian tube and tympanic cavity brings to one's attention several facts which may be of interest in the pathogenesis of this disease:

1. Eggston and Wolff[13] found that great variations in the size of the isthmus of the tube exist even in individuals of the same age. This may be one reason why some people develop tubal obstruction and effusion more readily than others under similar circumstances.

2. These authors state further that under normal conditions the tympanic mucosa consists of low cuboidal epithelium and the submucosa is often just thick enough to transport the relatively small blood vessels, nerves and lymph channels.

3. Eggston and Wolff have noted also that "the arteriolar walls are very delicate and frequently one can observe blood corpuscles in the act of escaping from the vascular channels at the time of fixation of the tissues."

Keeping the above facts in mind, one can readily see that the histologic structure of the middle ear is ideal for the effusion of serum in the presence of a vacuum; however, according to these same authorities, the mucosa is subject to "abrupt and marked changes under apparently slight stimulus," and by a combination of edema and actual multiplication of cells it may become several times its normal thickness.

The variability in the thickness of the mucosa and differences in capillary permeability are possible explanations why some middle ears develop an effusion and others do not.

The origin of the serous fluid in this disease is undoubtedly from transudation of fluid elements of blood serum through the thin walled arterioles and capillaries in the submucosa of the tympanic and mastoid cavities, but the origin of the "mucus type" fluid warrants more discussion. Politzer[2] and others have stated that it is the mucosal response to inflammation, but if the mucosa contains no secreting acinous glands one may wonder about the "secretory" origin of the fluid. Löwy,[14] too, was particularly concerned with the origin of the exudate. He believed that it must, of course, originate in the vessels, but was interested in determining if the cellular elements of the mucosa play an active part; that is, whether a true secretory process is involved. He proceeded on the premise that a secretory activity on the part of the epithelium should produce changes in the constituents of the serum and, on the other hand, if only an increase in permeability is involved, the exudate should retain the essential characteristics of the blood serum. He was able to demonstrate the identity of the blood group (type) in the middle ear exudate in each of 15 cases examined, whereas this proved impossible in the exudate of cases with suppurating otitis. He also proved by biopsy of the middle ear mucosa in a child with secretory catarrh (who had died of other causes) that severe mucosal edema existed. His conclusions were that a simple increase in the permeability of the capillaries favors the development of a serous otitis media and that his investigations do not indicate an active secretory activity on the part of the mucosal epithelium.

Lawson[8] states that "the mucus in the mucus type of the disease comes as a result of the closure of the Eustachian tube. It is the normal secretion of the mucous glands within the tube and may be increased temporarily to balance the external atmospheric pressure."

The presence of the mucus (not serum) in the middle ear, therefore, may be explained in one or both of two possible ways: *1.* because of blockage of the tube at its pharyngeal end, mucus secreted by acinous glands in the pharyngeal portion of the tube may have made its way by retrograde flow

into the tympanum; *2.* it may have been secreted by newly formed goblet cells in the tympanum. Quoting from Eggston and Wolff,[13] "No glands are present in the submucosa of the middle ear. Normally not even goblet cells are evident in the mucosa but in the presence of infection they may occur. How this tissue is kept moist is not known."

The evidence, then, favors the view that most of the fluid originates in the blood serum under the influence of negative pressure alone or by the added influence of inflammation and increased capillary permeability. It may be added to or replaced in some cases by the secretion of mucus by acinous glands in the Eustachian tube and by the goblet cells in the tube and tympanic cavity. The secretion by goblet cells in a metaplastic mucosa may explain the persistence of fluid in some ears after the Eustachian tube has become patent.

ETIOLOGY.

As has been mentioned in the preceding paragraphs, the primary factor in the etiology (of otitis media with effusion) is an obstructed Eustachian tube. Any condition which is capable of producing swelling of the tubal mucosa, compression of the ostium or lumen of the tube, or in any way interfering with the normal tubal function must be considered a possible etiologic factor. Robison[12] is of the opinion that the obstruction is often due to mechanical lymphatic blockage, secondary to inflammatory swelling of the lateral retropharyngeal lymph nodes which drain the sinuses and nasopharynx as well as the middle ear, resulting in retrograde lymph circulation and lymphedema of the tubal mucosa at the isthmus.

In addition to upper respiratory infections and lymphoid hyperplasias, there are many other local conditions which may be causative or predisposing factors. Allergy is an important and probably often overlooked cause, but on the other hand, we must be careful not to assume an allergic etiology merely because there is no other immediately obvious pathology. Dental malocclusion, tumors of the nasopharynx, and paralysis

of the tensor veli palatini muscle account for a small percentage of cases each. Marked septal deviations and cleft palate are predisposing factors.

Some of these patients have probably had a long standing partial obstruction of the tube because of a traumatic adenoidectomy or a previous suppurative otitis media. They tend to have a recurrence of serous effusion into the tympanum with each upper respiratory infection.

Most of us who have been disappointed with the results of our treatment have suspected for some time that obstruction of the Eustachian tube is not the only factor in the persistence of effusion into the middle ear. What else, then, may play a rôle in the persistence of the fluid? Hoople[1] has mentioned systemic conditions which abnormally affect tissue fluid balance: namely, allergy, cardiac insufficiency, cardiovascular-renal disease, and endocrine disturbances as potential sources of trouble, particularly in persistent chronic cases. These diseases probably affect the water balance by increasing the capillary permeability. Other conditions which increase capillary permeability may be important primary or secondary causes of effusion in the middle ear. Hilger[15] has pointed out that autonomic dysfunction, which is sometimes the result of psychic disturbance, may result in arteriolar spasm and capillary dilatation. Williams[16] has recently discussed the relationship of autonomic dysfunction to allergy. In an extensive review of the literature, he points out that anoxia in the involved tissue leads to increased capillary permeability and that "destruction of the leucocytes in the capillary loop together with more or less injury of other involved cells would result in the release of histamine, which also would increase capillary permeability." It seems reasonable that these effects of autonomic dysfunction could occur also in the middle ear and favor the continuance of an effusion in some cases.

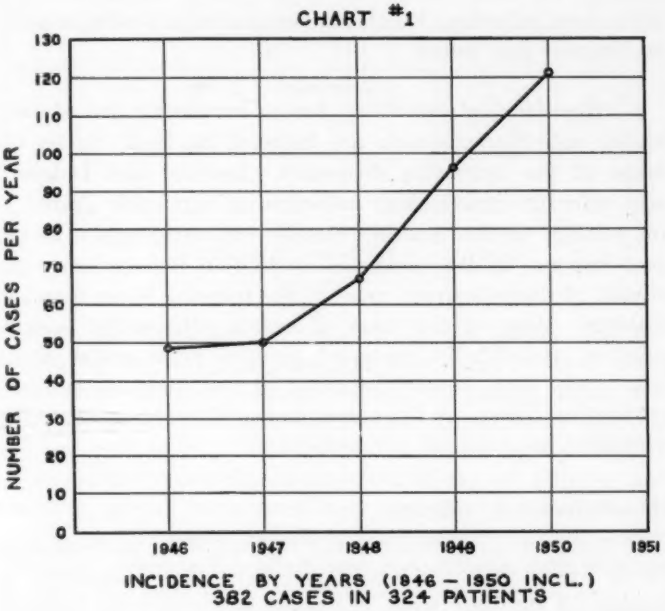
INCIDENCE.

I — Increasing in frequency?

That there has been an increase in the incidence of otitis media with effusion since 1940 is generally believed to be

true, but the reasons for the increase have not been agreed upon by various authors. The theories advanced to explain the greater incidence are as follows:

1. *Increased vigilance* on the part of otolaryngologists, once they have learned to recognize the condition, is believed to be the chief factor by Hoople[1] and also by Jordan.[17] This has certainly been true in my experience, as shown by the accompanying Chart 1. Prior to service in the Army, the author



recognized very few cases and since 1946 has observed a steady increase in incidence chiefly because he has looked for the disease.

2. *Virus infection.* Eagle[18] and others are of the opinion that many of these cases are due to viral infections in the

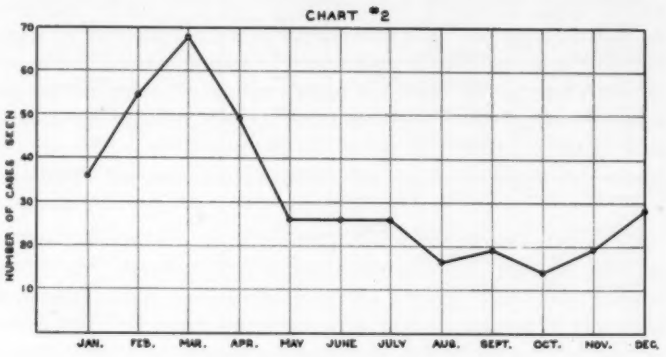
middle ear. The fact that the fluid is found sterile by ordinary culture methods does not, of course, preclude the possibility of a virus infection. Viruses inhabit tissue cells and are not found in the intercellular spaces or tissue fluids. Lawson,[8] while discussing the possible reasons for the sterile condition of the fluid, included the possibility that the investigative methods may be insufficient, that the organism may be ultramicroscopic, and further that the organisms may be in the mucosa but not in the exudate. Although Lawson did not mention the term "virus," all these criteria would hold true for a virus infection. Further investigation is needed to prove or disprove this theory.

3. Hypovirulent infections due to increasing use of antibiotics and chemotherapy are believed by Hall[19] to be the cause of the increasing frequency. Lawson[8] and Lederer[20] also refer to hypovirulent infection as a possible factor in the etiology of this disease. Hoople[1] has noted only a 20 per cent increase in the incidence of fluid in the ear since the advent of chemotherapy, most of the increase being found in children. Many of the cases of mucus otitis media seen in children undoubtedly represent aborted acute otitis media that would have become suppurative had they not received antibiotics. There are those, however, with mucus otitis media who have not received any medication.

II — Influence of Climate.

1. Temperature in itself is not believed to be an important factor except that the condition occurs most frequently in seasons of the year when the temperature is most variable and hence when upper respiratory infections are most prevalent. In the author's locality the largest number of cases were observed during the months of February, March and April (as shown in Chart 2), which happen to be months of extremely variable temperature. This has been observed also by others.

2. Altitude and humidity were first mentioned by Politzer,[2] and later by Ballenger,[5] Eagle[18] and Hantman,[11] as being possible influencing factors in the incidence of this disease. Their importance has been questioned by others. While re-



INCIDENCE BY MONTHS FOR 5 YEAR PERIOD
(1946 – 1950 INCL.)
382 CASES IN 324 PATIENTS

viewing the literature for this thesis, the author was impressed by the fact that most reports of large series of cases came from otolaryngologists residing in coastal areas and along river valleys where the altitude is relatively low and the humidity high. Well qualified otolaryngologists residing in localities where the reverse conditions of altitude and humidity prevail were questioned regarding the incidence of secretory otitis media, and their responses with one exception (Boulder) were to the effect that even though they were on the alert for such cases they saw only a few per month or a few per year.

Localities of reported high and low incidence with their respective ground elevations and relative average humidities are shown in Table 1. It is admitted that this survey is incomplete, but it seems reasonable to assume that an individual with pre-existing pathology in the Eustachian tube is

TABLE 1.

INCIDENCE IN VARIOUS LOCALITIES, SHOWING GROUND ELEVATION AND AVERAGE RELATIVE HUMIDITY.

Essayist or Reference	Location	Ground elevation above sea level(feet)	Average relative humidity	Incidence
Jones, C.C.[9]	Cincinnati,Ohio	533 ft.	79.2%	High
Hotchkiss, W.[22]	Miami,Fla.	8	77.2	High
Hall, S.S.[20]	Clarksburg,W.Va.	977	76.7	High
Robison, J.M.[12]	Houston, Texas	138	76.0	High
Parrior, J.B.[23]	Tampa, Fla.	24	75.5	High (Approx. 6 per week)
Mantman, Irvin[11]	Keesler Field,Miss.	9	74.7	High
Shaninian, L.[24]	San Francisco,Cal.	52	73.6	High
Eagle, Watt[19]	Durham, N.C.	406	72.0	High
Hoople, Gordon[1]	Syracuse, N.Y.	424	71.7	High (3-4% of total pts.)
Lindsay, John[25]	Chicago, Ill.	611	71.7	High
Author		621	69.5	High
McDougall, C.[26]	Atlanta, Ga.	977	69.2	High
Jordan, May[18]	Pittsburg, Pa.	1248	68.5	High
Hickey, H.L.[27]	Denver, Colo.	5292	52.7	Very low (Approx. 6 per yr.)
Bradley, J.W.[28]	Colorado Springs, Colo.	6175	50.7	Low except in Spring months then 6-8/mo.
Lamberson,W.H.[29]	Colorado Springs, Colo.	6175	50.7	Low
Richardson,Geo.[30]	Albuquerque,N.M.	5310	46.2	Very low Almost nil
Cruthirds, A.B.[31]	Phoenix, Ariz.	1083	41.7	Very low (3 per 500 pts
Spearman, M.P.[32]	El Paso, Tex.	3936	40.2	Extremely low. Almost nil.
Page, Donald[33]	Boulder, Colo.	5400	35-40?	Low except in Winter

more likely to develop tubal obstruction and effusion in a moist climate at or near sea level than he would be in a high dry climate.

III — Age Incidence.

Secretory otitis media may occur at any age but is most prevalent in young children, undoubtedly because they have more upper respiratory infections than any other age group, and because they are predisposed to tubal obstruction by enlarged adenoids or by hypertrophied lymphoid tissue in the fossa of Rosenmüller and furthermore by the smaller size of the pharyngeal opening of their Eustachian tubes as pointed out by Tremble.[33] In the author's series, 120 of the total or almost one-third of the patients were children under 10 years of age. The examination of many of these children was prompted by impaired hearing as discovered during school hearing surveys.

For convenience of discussion, the author's series of 372 patients was divided into acute, subacute and chronic groups, depending upon the duration of the attack. The cases termed acute were of short duration with disappearance of the fluid within less than three weeks from the onset of symptoms. The subacute cases persisted from three to six weeks, and those termed chronic continued to have fluid in the middle ear for a period of six weeks to several years. One patient with malocclusion gave a history of having had symptoms for 10 years.

The various causes for each group are shown in Table 2. Many of these patients had more than one etiologic factor but only the predominant one was selected. When active nasal allergy persisted, it was listed as the primary cause.

In the author's experience, an upper respiratory infection was the exciting cause in almost two-thirds of the cases. Allergy was considered to be the etiologic factor in 37 or approximately 10 per cent of the total number and 11 per cent of the chronic cases, even though allergy was constantly kept in mind and searched for. Nasal smears for eosinophiles were done almost routinely, and skin tests were done on many of those with questionable signs of allergy. The author has not observed any appreciable increase during the ragweed hay fever season, which is very prevalent in his locality from late August to October.

In children, hypertrophied adenoids or hypertrophied remnants of lymphoid tissue in the fossa of Rosenmüller were the chief predisposing factors. The majority of these chil-

TABLE 2.
ETIOLOGY IN 372 CASES.

ETIOLOGY	Acute	Subacute	Chronic	Total
Acute U.R.I.	111	35	16	162
Sinusitis	24	16	19	59
Hypert. adenoids (without apparent allergy)	7	20	37	64
Allergy	19	7	11	37
Previous Supp. O.M. only cause	0	0	3	3
Aero-otitis	9	1	0	10
Contagious disease (measles, mumps)	2	0	0	2
Pregnancy (Endocrine)	1	0	0	1
Hodgkin's disease	0	0	1	1
Malocclusion	0	2	3	5
Malignancy of nasopharynx	0	0	2	2
Swimming	3	0	0	3
Unknown	7	9	7	23
	183	90	99	372

dren developed bilateral effusion with each cold. The greatest lymphoid hypertrophy occurred in the allergic children, as was emphasized by Jordan,[17] but the nonallergic children with large adenoids far outnumbered the allergic ones insofar as

could be determined. It is admitted that there were very probably a number of patients whose obscure allergies were not detected.

Among adults, infections of the paranasal sinuses were very prevalent. Most of these had involvement of one or more sinuses on the homolateral side, but curiously enough a small number had involvement only on the ipsilateral side. Malocclusion was the only etiologic factor in five, and lymphoepithelioma of the nasopharynx was the cause in two patients, one of these having bilateral effusion.

Of interest was the unfortunate fact that the etiology could not be determined in 23 of the total. Seven of these ran a chronic course.

PATHOLOGY.

As mentioned above, there are two clinical types of this condition, serous and mucus, but many cases constitute a combination of the two.

The serous fluid is thin, clear, light yellow and watery when first evacuated or aspirated from the tympanum, but becomes thicker and coagulates when exposed to air. It is basically a transudate containing few inflammatory cells and usually has a low protein content, although the protein is quite variable.

The so-called mucus fluid is thicker and tenacious, sometimes turbid, usually very viscid, and much more difficult to remove. It contains many inflammatory cells, mostly polys, and usually has a high protein content. It is, therefore, an exudate. Both are sterile by ordinary culture methods, as shown by Forschner[34] and others.

CYTOLOGY.

During the past year the author has almost routinely examined smears of the ear fluid stained with Hansel's stain and has observed that the number of inflammatory cells per high power field varies tremendously. The thin serous fluid in cases without associated inflammatory disease contains only

a few cells per H.P.F., whereas the more tenacious fluid may contain as many as 300/H.P.F. or more, predominantly polys.

I have been particularly interested in whether or not eosinophiles occur in the ear fluid of those patients whose secretory otitis is believed to be the result of allergy. Koch[25] refers to Dohlman's report in 1941-1942, wherein the latter found eosinophiles in the secretion of secretory middle ear catarrhs, chronic otitis and mildly acute otitis, "in certain cases in a very large number," and states that Dohlman considered these to be of allergic origin. He states further that Dohlman has given merely a general survey of his experiences, without any statistics with regard to percentage of eosinophiles found in the secretory otitis medias. Hotchkiss[21] observed that "even in definitely allergic cases, the eosinophilic count is not dependable." The only other published report on the incidence of eosinophiles in secretory otitis media that the author could find was that by Jordan,[17] who stated that he found "occasional eosinophiles" in direct smears of the fluid, and 73 per cent of his chronic cases were attributed to allergy.

I have looked painstakingly for eosinophiles in the ear fluid of at least 50 patients, including all those who had evidence of nasal allergy and those in whom the etiologic factor was assumed to be allergy, and have yet to find an eosinophilia in a single case; furthermore, I have seen only *one eosinophile* in the total of 50 slides examined! I do not mean to infer by this, however, that an absence of eosinophiles in the ear fluid precludes an etiologic diagnosis of allergy as the cause of tubal obstruction. The allergic reaction may be limited to allergic swelling in the pharyngeal end of the tube, thereby creating a negative pressure, without producing a true allergic edema and eosinophilia in the tympanum.

CHEMISTRY OF EAR FLUID.

Löwy[14] states that Schlander and Goebel demonstrated by means of protein determination on the middle ear exudate that the fluid is due partially to an exudation and partially to a transudation, and that the majority of the secretory

catarrhs can have only an inflammatory origin, but their manuscript is not available for review. There is disagreement among other authors concerning which type of fluid has the highest protein content, the thin serous type or the thick mucus exudate. Robison[12] found a high total protein content in the exudate containing many polymorphonuclear leucocytes and a low protein content in the thin transudative fluid containing a few polys. Hoople,[1] on the other hand, states that the thin serous fluid has a high protein, and the thick mucus exudate a low protein content.

According to Cantarrow and Trumper,[36] the protein content of transudates and exudates in various parts of the body can vary tremendously because of variations in the intensity of the inflammatory process and variations in capillary permeability. They state that in the case of exudates resulting from inflammatory processes of lesser intensity, such as pneumonic pleurisy and tuberculous peritonitis, the total protein concentration usually ranges from 0.1 to 5 gm. per 100 cc. The protein content of noninflammatory edema fluids or transudates is said to be relatively low (0.1 to 1 gm. per 100 cc.), since their pathogenesis is usually independent of alterations in capillary permeability. They state further, however, that if the effusion has existed for some time, water may be reabsorbed more rapidly than solids, resulting in a slowly increasing concentration of protein which may eventually approach that of true exudates.

I have, through curiosity, done some protein determinations along with cytologic smears in a small number of cases to see whether there is any constant correlation between the total protein and the number of inflammatory cells in the fluid, which is what one would normally expect.

The fluid was collected by aspiration through the drum with a short bevelled 20 or 22 gauge needle attached to a tuberculin syringe. The volume of fluid obtained by this method varied from 0.1 to 0.4 cc., but usually only 0.1 to 0.2 cc. could be collected. The fluid was diluted to 2 cc. with normal saline and protein determination done in the usual manner as for serum protein.

The results, along with the cell count, gross description of the fluid, the etiology and chronicity are shown in Table 3.

TABLE 3.

Patient	Gross appearance of fluid	Protein content	Cell count	Etiology	Chronicity before trt.	Result
#1. M.P.P.	Thin serous	1.25 Gms/ 100 cc.	Not done	Cold	1 week	Cleared in 1 week
#2. J.P.K.	Thin serous	1.45 Gms.	22/HPF most polys	?	1 month	Fluid persists after 3½ mos.
#3. J.S.	Thin watery light yellow serous	3.4 Gms.	18/HPF	Nasal allergy	1 day	Cleared in 1 week
#4. O.H.R.	Thin watery light yellow serous	3.9 Gms.	No inflam. cells. Few rbc/HPF	Partial cicatricial stenosis of E.T.? Nasal smear neg.	4 months this attack. Recurrent since 1920.	No follow-up
#5. H.L.M.	Med. consistency serous	4.4 Gms.	25/HPF Predom. lymphocytes plus 27rbc/ HPF	Supp. max. sinusitis, bilat.	2 weeks	Cleared in 3 weeks
#6. J.L.H.	Thick mucus glue-like	5.0 Gms.	225/HPF Mostly polys	Cold plus narrowed eust. orifice Nasal smear neg.	3 weeks Hist. of similar attack 10 yrs. before	Fluid persists after 8 mos. Cleared for periods of 6 wks. and 8 wks. Recurred with colds.
#7. C.S.	Thin serous	5.25 Gms.	No inflam. cells. Few epithelial cells.	Hodgkin's disease. Lymphatic blockage?	2 months	Cleared 1 mo. after external irradiation.
#8. G.C.	Medium consistency serous	6.2 Gms.	26/HPF plus large clumps.	Cold. Nasal smear neg.	6 months	No recurrence.
#9. A.E.J.	Medium consistency serous	7.2 Gms.	2/HPF plus 10-15 rbc/ HPF	Cold. Nasal smears neg.	3 months	No recurrence.

As shown by a study of the above table, there was no constant parallel between the total protein content and the cell count, although the thin, watery, serous fluid tended to have a lower protein and a lower cell count than the more tenacious fluid. If only two of these were selected for comparison, however, *e.g.*, No. 6, J. L. H., who had a thick mucus type of fluid with 225 polys/H.P.F., definitely an exudate, and No. 7, C. S., who had a thin serous type of fluid containing no inflammatory cells and without question a transudate, one might come to the conclusion that the protein content of the mucus type is lower than that of the serous type, which is probably erroneous in most cases.

The percentage of error due to the dilution factor is estimated to be only approximately 10 per cent. The protein content may be affected very slightly by the presence of a few erythrocytes, but none of the fluid was grossly blood tinged; and I know of no less traumatic method of collecting the fluid than with a needle and syringe. The thick mucus fluid is very difficult to collect by any method.

The only conclusion that can be drawn from this study is that a larger number of determinations should be carried out to see whether any correlation can be shown between the gross appearance of the fluid, cell count, protein content, duration of the disease, etc. The author intends to continue this investigation according to this plan.

SYMPTOMS.

Adults with an accumulation of fluid in the middle ear usually describe their sensations realistically. Most complain of a "stopped up" or "full feeling" in the ear. Others state quite accurately that they have water in the ear and at times they can feel fluid moving about or bubbling upon sniffing or forcefully blowing the nose. Many will say that the ear feels numb or "dead," and this sensation of numbness is imparted also to the skin around the auricle, noticeable upon rubbing the ear or combing the hair on the affected side. These patients usually have a full tympanum and mastoid cavity.

Autophony is commonly described as "talking with the head in a barrel." A diagnostic symptom, if the middle ear is only partially filled, is the temporary improvement in hearing upon tilting the head toward the affected side or upon lying down, thus permitting the fluid to drain away from the round window, or probably because the pressure of the fluid upon the round window is relieved thereby.

They may give a history of having been treated by other otolaryngologists by inflation with improvement in hearing for a short time.

In the case of young children, the only symptom is usually an impairment of hearing which has been noticed by the parents, the school teacher, or after routine school audiograms.

DIAGNOSIS.

The diagnosis can usually be suspected from the history. Inspection usually reveals the characteristic amber or yellow color imparted to the translucent drum, which is not inflamed and may or may not be retracted. If the drum is opaque, this amber color usually cannot be detected. In long standing cases the drum occasionally has a bluish color, probably due to the congested mucosa on the promontory. Hoople[1] has emphasized the chalk white appearance of the malleus handle and short process which gives the impression that the drum is retracted when it may not be at all.

If the tympanum is not filled with fluid, one can often see a fluid line or bubbles through the drum, and when these signs are present the diagnosis is readily apparent; however, a full tympanum is encountered many times more frequently than the drum with a meniscus. The pneumatic otoscope or Politzerization may be helpful in demonstrating bubbles or a fluid line.

The hearing loss, conductive in type, varies with the amount of fluid present. The audiometric curve, although not characteristic, is most often flat unless there is a pre-existing inner ear lesion. Löwy[37] observed that in approximately one-third of the cases, the hearing loss was severe, with consider-

able loss in the range of the higher tones. He does not attribute this to damage of the inner ear, but to increased tension and impaired mobility of the fenestra rotunda, as the signs of inner ear impairment largely disappear following evacuation of the fluid.

Wever[38] recently commented upon the effect of fixation of the ossicular chain by fluid, saying, "When it comes in contact with the drum membrane and ossicles this substance (fluid and semifluid substance) dampens and loads their movements, and when finally it fills the cavity it adds stiffness as well. The result is an impairment of sensitivity that in the early stage affects the high tones the most seriously, and then in the severe stages includes all tones."

The next step usually recommended, if one is not certain of the presence of fluid, is the diagnostic inflation using a catheter and diagnostic tube. Many auscultatory sounds have been described in detail by Hoople,[1] Hotchkiss[21] and others, and need not be repeated here. As one becomes more accustomed to recognizing the drum with fluid behind it, the necessity for diagnostic inflation diminishes, unless, of course, the drum is opaque. The author is in agreement with Eagle[18] that it is preferable to avoid using the Eustachian catheter if at all possible because of its traumatic effect upon the pharyngeal orifice of the tube and the risk of prolonging the disease.

If one suspects fluid in the tympanum, a diagnostic paracentesis may as well be done initially if the symptoms justify it, the indications being the same, of course, as though one had no doubt about fluid being present. This can be done without anesthesia with a short bevelled needle attached to a tuberculin syringe, or in the usual manner with a knife. If fluid is encountered upon suction or Politzerization, the opening must be enlarged as for treatment purposes, particularly if the fluid is of the thick mucus type. One can usually foretell the nature of the fluid by the appearance of the drum. When the fluid is serous, the drum is more apt to be thin and transparent, with an amber or yellow color. When thick

mucus is present the drum has a more reddish hue with a dispersed light reflex, *i.e.*, more of a sheen on the surface, and it is less transparent.

Having established the presence of an effusion in the ear, one must then search carefully for its cause. If the etiology is not apparent in the nose, sinuses, mouth, nasopharynx or neck, one should enlist the aid of an internist who is in a better position to discover possible systemic disorders.

Cytologic study of smears of the ear fluid will often give a clue as to whether the etiology is inflammatory or noninflammatory by the number of polymorphonuclear cells present.

PROGNOSIS.

With regard to prognosis, most observers agree that the longer the condition has persisted prior to treatment, the less favorable the chances for a complete cure.

Ears containing a small amount of fluid respond to treatment more promptly than those which are full. The mucus type of secretory otitis media is more often resistant to treatment than the serous type, probably because there is a low-grade inflammation of the mucous membrane of the Eustachian tube and tympanum, and these changes become irreversible after a variable period of time. Those patients with a blue discoloration of the drum are usually more resistant to treatment. Patients in whom one is unable to find any definite etiologic factor have much less chance of getting well, although they may do so spontaneously if and when the unknown pathology subsides.

Why some ears develop adhesive processes after a relatively short time and others continue to harbor fluid for many years without forming adhesions is not known. We do know that the fluid coagulates readily when exposed to air, and it may be that when fluid remains in the tympanum after the Eustachian tube becomes patent, it then coagulates and tends to form adhesions. We should, therefore, continue treatment as long as any fluid remains in the middle ear.

In the vast majority of cases, the hearing returns to normal after the fluid is evacuated, regardless of the number of myringotomies, unless there are adhesive processes or preexisting inner ear pathology.

TREATMENT.

There are two objectives in the treatment of otitis media with effusion; namely, to rid the middle ear of fluid and to prevent its recurrence by treating the associated etiologic condition if this can be determined. The preservation of normal hearing is the most important objective, but this will be attained if the ear is kept free of fluid and adhesive processes are thereby prevented.

The first objective is much more easily accomplished than the second, and unless we are able to make a correct etiologic diagnosis we are almost sure to fail except in the acute cases of recent onset, which have a fair chance of clearing up spontaneously.

The treatment varies somewhat with the duration of symptoms, the quantity and type of fluid present, and the cause of the tubal obstruction.

Cases with symptoms of short duration, such as an aerootitis media with effusion, or those with an associated acute inflammatory condition in the nose or throat will often subside after proper attention to the acute nasopharyngitis or sinusitis, perhaps combined with subsequent Politzerization after the nose and nasopharynx are free of pus.

In the absence of a coexisting acute inflammatory condition in the nose or throat, if there is only a small amount of fluid present and the hearing is not markedly impaired, one is usually content with trying Politzerization alone for a week or so, but at the same time searching for and treating the cause of the tubal obstruction.

If the tympanum is full of fluid, as is the case more often than not, or if the history indicates that the fluid has been present for a week or longer, one may as well proceed with

myringotomy at the first visit, except in children with nasal allergy or infected hypertrophied adenoids. These children's ears will clear up only when the allergy is controlled or the obstructing lymphoid tissue removed by one method or another.

The myringotomy incision is usually made along the inferior half of the drum, and as long as possible, with the hope that it will remain open for a week or so; however, it usually heals within 48 hours, which is much too soon. The myringotomy is then repeated as often as the fluid accumulates.

Having opened the drum, we have a choice of several methods of forcing the fluid from the middle ear; namely, Politzerization, catheter inflation, suction applied to the external canal with Siegel's otoscope, spot suction, or a combination of these. The author has learned by experience, as did Politzer,[2] Eagle.[18] Hotchkiss[21] and others, that catheterization inflicts too much trauma on the orifice of the Eustachian tube and probably prolongs many cases that might have cleared up promptly had the catheter not been used. I have seen several patients with symptoms of brief duration and with little or no visible swelling about the orifice of the tube on first examination, who, after several catheterizations, were observed to have definite edema of the orifice and then ran a protracted course. Since this observation was made, I have used Politzerization and suction exclusively to remove fluid after myringotomy. Catheterization for auscultation is sometimes necessary to establish a diagnosis of fluid in the ear, in which case the beak of the catheter should be placed gently under direct vision with a nasopharyngoscope.

If the fluid is of the thick mucus type, the incision in the drum is made longer and spot suction is applied simultaneously with Politzerization to prevent the mucus mass from being drawn back into the tympanum as soon as the positive pressure has ceased and the intratympanic negative pressure again prevails. The placing of a pledget of cotton against the myringotomy opening to absorb the serous and seromucoid fluid as it is forced out also aids in evacuating the fluid

more effectively. After one has emptied the tympanic cavity as well as is deemed possible at one sitting, a pledget of cotton left in place against the drum will usually absorb additional serous fluid as it subsequently drains from the mastoid cells into the tympanum.

Having evacuated the fluid from the middle ear, one must not forget to treat the primary condition responsible for its presence. Local conditions in the nose and nasopharynx which might act as influences toward recurrence should be corrected.

Jordan[17] employs a trial period on antihistaminics if the etiology is not established and when allergy is suspected.

Jones[9] reported good results with the use of an autogenous vaccine made from culture of the nasopharynx in a few chronic recurrent cases. The author has had no personal experience with vaccines in the treatment of this disease.

The use of a permanent perforation in the drum has been advocated by some and condemned by others. Hotchkiss[21] believes that it is justified in selected cases which do not subside in spite of corrected causative disease, especially in older individuals in whom a perforated drum is of little concern. (He uses a corneal trephine on a long handle to make the permanent opening.)

Mastoidectomy has been performed as a last resort by several authors in chronic resistant cases. Although this writer has performed a mastoidectomy on only one patient with secretory otitis media, he has formulated the following opinion regarding the type of case most suitable for operation: *1.* a chronic secretory otitis media resistant to all other forms of treatment, *2.* with corrected or with no apparent associated nasopharyngeal or sinus disease, *3.* with an inflatable Eustachian tube, *4.* responding temporarily to myringotomy and inflation, indicating that the hearing loss is not irreversible, and *5.* with a fairly well pneumatized mastoid. Conversely, there would be little or nothing to be gained by operating upon a sclerotic mastoid, upon a patient whose Eustachian

tube is noninflatable, or upon a patient whose hearing does not return to a useful level at least temporarily after evacuating the fluid.

RESULTS OF TREATMENT.

Considering the series as a whole, the results of treatment were fairly satisfactory. As mentioned earlier in this paper, there were 183 patients whose symptoms lasted from a few days to three weeks, 90 patients who were well after three to six weeks, and 99 whose disease persisted longer than six weeks. The total of 372 patients had a total of 439 separate attacks. Forty-six patients had from one to 16 recurrences.

Although a total of 449 myringotomies were done, 104 patients in the acute and subacute groups became rid of the fluid without myringotomy. This number does not include 28 children in all categories whose ears returned to normal without myringotomy following adenotonsillectomy. Another 115 patients in the acute and subacute groups returned to normal after only one myringotomy; 31.2 per cent of the total had bilateral involvement.

Considering only the group of 99 chronic cases, the results were more disappointing. Of these, 52 were eventually declared well and 18 improved. Another 18 were not improved after prolonged treatment, and there was insufficient follow-up observation in 11 patients. Two of those not improved had lymphoepithelioma of the nasopharynx.

The results in 45 patients in the chronic group treated by various methods for hypertrophied lymphoid tissue about the Eustachian orifice are shown in Table 4.

The author discontinued using the radium applicator several years ago, but it seems that the immediate results in this small group were essentially the same as with external irradiation. Several of those who did not respond to irradiation were later found to have an allergy as the primary etiology.

Of the 11 patients with chronic secretory otitis of allergic etiology, four became free of fluid while receiving injections

of dust and mold extract, three improved on allergic management before they discontinued treatment, and two showed no improvement with antihistaminics or specific therapy. The remaining two patients in this group were of interest in that they may well be classified as belonging in the category of

TABLE 4.

RESULTS OF TREATING HYPERTROPHIED LYMPHOID TISSUE IN NASOPHARYNX, IN 45 CHRONIC CASES.

Method of Treatment	Total no. treated	Number cured	Not cured
Radium applicator	15	8	7
External irradiation	9	5	4
Adeno-tonsillectomy	22	19	3
	46*	32	14

*One of those not improved after adenotonsillectomy later cleared up after external irradiation.

intrinsic allergy with perhaps an autonomic dysfunction and arteriolar spasm as the underlying pathology. In any case, they both responded to vasodilator drugs after being resistant to all other forms of treatment. Their case histories are summarized as follows:

Case 1: Mrs. L. W., age 51. Complained of roaring and fullness in left ear, vertigo, and throbbing pains in left temporal and parietal regions daily for two weeks following a "cold." The left drum was retracted and amber in color. Rinne negative. Serous fluid was evacuated on myringotomy and inflation. The nose, sinuses, nasopharynx and Eustachian orifice appeared essentially normal. The fluid recurred after a few days when myringotomy and inflation was repeated and an antihistaminic prescribed. She continued to have recurrences of fluid and left-sided headache, and during the ensuing six weeks, five myringotomies were done. An X-ray of the mastoid revealed "moderate increase in density of the air cells of the left mastoid with some haziness of the cell walls. No bone destruction. Findings are consistent with an acute or subacute mastoiditis, left." The X-ray changes were, of course, due to the presence of serous fluid in the mastoid air cells. Nasal smears were negative for eosinophiles. Two months after the initial visit she still had fluid in the ear and almost daily attacks of left-sided headache which seemed to be similar to histamine cephalgia. She was given daily subcutaneous injections of 1:100,000 histamine and after four days the headache did not recur. The fluid in the left ear was blown out once more at this time. Histamine injections were continued and subsequent examinations over a period of two months showed no recurrence of effusion in the middle ear. Hearing returned to 15/15 for whispered voice.

Case 2: Mrs. J. F. K., age 49. c.c. Fullness in both ears and impaired hearing for 10 days. Two inflations elsewhere with very temporary improvement. History of occasional hay fever in the past but no associated symptoms at time of onset of ear trouble. Both drums retracted with serous fluid in tympanic cavities. Eustachian orifices edematous. Nasal mucosa slightly paler than normal, sinuses and nasopharynx negative. Nasal smear and ear fluid negative for eosinophiles. W.B.C. normal. Pyribenzamine was prescribed empirically. On Politzerization air traversed the right Eustachian tube but not the left. During the following two and one-half months the drums were incised many times and fluid aspirated, but each time the ears became full again within a few days. The Eustachian orifices remained slightly edematous. Further investigations had been carried out, including skin tests which were all negative. The B.M.R. was minus 17 and the blood cholesterol was very slightly elevated. On the strength of this, thyroid extract was prescribed by the internist without improvement. A food allergy was then suspected but there was no improvement on a nonallergic diet. A culture of the nasopharynx revealed hemolytic streptococci and an autogenous vaccine was ordered, but never used. While the vaccine was being prepared several bits of tissue were removed from the nasopharynx for biopsy that were negative for malignancy. Then one day the patient presented herself with an edematous swelling of the left upper eyelid. This suggested the possibility of arteriolar spasm and capillary dilatation as the cause of the edema. She was started on niacin 100 mgm. t.i.d. before meals. Both drums were opened, with evacuation of a large quantity of serous fluid. There has been no recurrence of fluid for a period of six weeks.

Both patients had associated signs of vasomotor or autonomic dysfunction — a vascular headache on the homolateral side in Case 1, and an edema of the eyelid and a history of extreme sensitivity to temperature changes in Case 2. The response to the histamine desensitization and to the niacin may have been coincidental, but the evidence seems more to the contrary. A longer period of observation is needed before any definite conclusions can be drawn.

SUMMARY AND CONCLUSIONS.

1. The available literature pertaining to the subject of secretory otitis media has been reviewed and the author's series of 372 cases analyzed with regard to etiology, pathology and treatment.

2. The apparent increasing frequency of this disease is largely due to increased vigilance on the part of otolaryngologists during the past decade, and to a lesser extent due to the influence of widespread use of antibiotics. The rôle of virus infections (in the etiology of this disease) has not been satisfactorily determined.

3. A scattered survey of the incidence in various parts of the country suggests that secretory otitis media occurs much more frequently in areas of low altitude with a high relative humidity.

4. Upper respiratory infections are the chief predisposing causes in adults as well as in children. Hypertrophied lymphoid tissue in the nasopharynx plays an important rôle in children. Allergy accounted for 10 per cent of the entire series and 11 per cent of the chronic cases.

5. Differences in the thickness of the tympanic mucosa and variations in capillary permeability may be deciding factors as to which ears with an obstructed Eustachian tube develop effusion and which ears do not. Autonomic dysfunction can adversely affect capillary permeability.

6. Normal variations in the size of the isthmus of the Eustachian tube may explain in part why some ears become chronic and others do not under similar circumstances.

7. Some middle ears continue to harbor fluid after the Eustachian tube has become patent. The secretion in these ears is usually of the mucus type, the result of continued low-grade inflammation of the mucosa which probably has undergone metaplasia.

8. Protein determinations on the ear fluid of a limited number of the author's patients does not support the view that the serous fluid has a high protein content or that the mucus fluid has a low protein content. Wide variations were found to exist in similar types of fluid. Comparative studies of the gross appearance of the fluid, the cell count, and protein content showed that thin serous fluid usually had a low cell count and low total protein; however, no definite pattern could be established. Further comparative study of a larger number of such determinations is indicated.

9. Eosinophiles rarely occur in the fluid of secretory otitis media, even when the tubal obstruction is the result of allergy. An eosinophilia was not observed in any of the more than 50 slides examined.

10. The Eustachian catheter should be used as gently and as sparingly as possible in the diagnosis and treatment of this disease as the trauma inflicted on the pharyngeal orifice of the tube undoubtedly prolongs the course of the condition.

11. Mastoidectomy should probably be done more often on the chronic resistant cases to preserve hearing. The author has expressed his opinion as to the type of case best suited for mastoidectomy.

12. The vasodilator drugs may prove useful in the treatment of this condition if there are associated signs of autonomic disorder. Two cases are cited in which they were seemingly of therapeutic value.

13. Secretory otitis media remains one of the greatest problems in otolaryngology today, but this fact is still not appreciated by many.

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NASAL SINUS DRAINAGE.
Factors Which Influence Bernoulli Action.*

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INTRODUCTION.

The author has written three previous articles on Bernoulli action in the nose in which he has attempted to show various mechanisms whereby blowing of the nose produces suction at the sinus ostia.[1-3] The present paper is a discussion of various factors which influence Bernoulli action.

We should hold in mind throughout this discussion that we are not considering air currents in normal or even in forced respiration; nor are we concerned with the atrophic nose or even with a normal nose with wide breathing space. At the moment, we have no interest in what pattern a puff of smoke will make in the nose of a cadaver.

Our remarks at present are strictly limited to *forceful expirations* through a *congested nose.* There is another fact which we should keep in mind. The constriction of the air space of the nose to which we shall refer is caused by swelling of the turbinates. The turbinates are composed of erectile tissue and erectile tissue in the nose is just as unpredictable and irresponsible as erectile tissue found elsewhere in the body. This explains why the Bernoulli effect is so variable when we blow our noses. Thus, when recovering from a head cold, one may blow his nose without result and a few seconds later blow it with very satisfying productivity.

I — Conformation of Residual Air Space in Nose.

If the nose is congested to such an extent that there is only a small air passage through it, we are interested only in the

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shape of that residual air passage. If this remaining air passage is comparatively straight and is of an equal caliber or bore throughout its length, we can expect no Bernoulli effect. On the other hand, if this air passage is constricted anywhere along its entire length, there will be increased velocity of air at the constriction and diminished pressure in that area when one blows his nose. If a nasal sinus ostium is located in the area of constriction of this residual air passage, it is subjected to Bernoulli action when one blows his nose.

It is difficult to conceive of a conformation of this residual air space which can produce suction simultaneously at all ostia which open into it. It is equally difficult to imagine a constricted air space which does not produce suction at at least one ostium along the line.

II — Residual Air Space in Sinuses.

Throughout this paper, I have intentionally avoided frequent use of the term "suction" as applied to Bernoulli action. This was done in an effort to have us keep in mind that the only force which can be employed in forcing mucus through the ostia into the air stream is the pressure of the air in the sinuses. Regardless of the actual air capacity of a given sinus, this air capacity may be encroached upon by swelling of the soft tissues within the sinus and by the accumulation of mucus or pus within the sinus. So, in a nasal sinus, the only effective air capacity is the residual air space.

Let us assume, as we did above, that the ostium opens into a constricted portion of the residual nasal air passage into an area of diminished pressure. On blowing the nose, we start off with approximately atmospheric pressure of the air in the sinus, which pressure rapidly diminishes as the mucus is forced through the ostium. This motion of mucus continues until the pressure within the sinus is no greater than the pressure in the nasal air passages or until the mucus bubble bursts. On the termination of the blow, atmospheric pressure re-establishes in the sinus. When mucus is again condensed at the sinus ostium by ciliary action, we have proper conditions for a repetition of the process.

III — Patency of Sinus Ostia.

It stands to reason that, if a sinus ostium is solidly blocked by soft tissue swelling, it is incapable of transmitting any Bernoulli effect to its sinus. This is true whether the blockage is in the ostium proper or whether the obstruction is caused by overlying turbinate edema. It is possible, however, for soft tissue to overlie the ostium in such a manner as to block its normal opening into the air space but allow it to communicate with the residual air passage of the nose at a more proximal or distal position. In such a case, this fissure or slit serves as an adventitious ostium and, if it is located in an area of air space constriction, it will act like a true ostium as a pathway for Bernoulli action.

If an ostium is blocked with mucus, it does not preclude Bernoulli action. As we shall discuss later, such a condition is ideal for productive Bernoulli effect.

IV — Viscosity of Mucus.

The mucus can be so thick that there is difficulty in forcing it through the ostium. On the other hand, it can be so watery that the cilia cannot condense it at the sinus openings.

In the first case, more force than usual is required to force the mucus into the air passage. In the latter instance, Bernoulli action is ineffective for the simple reason that it cannot grasp the mucus to remove it.

V — The Force Which One Employs on Blowing His Nose.

Some years ago, when I was afraid that some Bernoulli critic would object to my drawn glass tubes which I used to represent constrictions in the nasal air passages, I resorted to a straight glass tube with a tap in the middle to represent a sinus ostium.

Into the straight tube I inserted two rubber dam tubings filled with water. The ends of this tubing were pulled together on the outside of the glass tube and tied in position on either side of the tap. The rubber tubing inside the glass tube,

being distended with water, encroached on the air space and fairly represented a nasal constriction formed by apposition of turgescent turbinates.

I attached a water manometer to the tap and blew through the glass tube. I found that when I blew lightly there was a slight amount of suction, but when I blew forcefully, the manometer registered pressure. This puzzled me for a long time until it was finally explained to me by one of my engineer friends who noted that when I blew with force through the tube the soft rubber and water constriction moved distally.

Thus, it is conceivable that on blowing the nose one may get suction at a given sinus ostium when he blows lightly and get no suction at all when he blows harder because of the fact that he distorts the congested turbinates and moves the area of constriction anteriorly.

The opposite effect of varying air pressure on blowing the nose is illustrated by the following experiment:

Push a close fitting rubber tube into a straght glass tube which has one middle tap. Adjust the rubber tube so that it comes up close to the tap. Blow gently through the rubber tube. A manometer attached to the tap shows suction.

Continuing to blow lightly through the rubber tube, withdraw it gradually until no suction registers on the manometer. Now, holding the tube in the same position, blow forcefully. Again the manometer registers suction.

The change in Bernoulli effect due to the force exerted probably explains the fact that an accomplished nose blower has a repertoire which varies from a light zephyr to a trumpeting blast.

If the constriction is distal to the ostium, reversal of the air flow by sniffing may produce effective Bernoulli action.

VI — Condensation of Mucus at Sinus Ostium by Ciliary Action.

We will take as an example one of the maxillary antra which has accumulated a considerable amount of mucopus on its

floor. We will assume that its ostium opens into the middle meatus at the most constricted portion of the residual air space of a congested nose. The ostium is patent and mucus on the floor of the antrum is of the ideal viscosity for forcing through the ostium.

We might think, with the above mentioned favorable factors, that all conditions will have been met for a productive nose blow; but there has been no condensation of mucus at the sinus ostium, and the blowing of the nose will result only in a momentary rarefaction of the air in the sinus and will be entirely unproductive.

How can the mucus be brought to the ostium? I know of only four mechanisms by which this can be accomplished: *1.* we might tilt the head so that mucus reaches the ostium by gravity; *2.* the antral mucous membrane may swell and bring the mucus up to the level of the ostium; *3.* we may have increased secretion which brings the level of mucus to the ostium; *4.* the cilia of the antral lining may walk the mucus along the floor and walls to the sinus opening. Certainly there is no disagreement in the opinion that ciliary action is the most important mechanism for transporting mucus and condensing it at the sinus ostium. This condensation is an absolute requisite for productive Bernoulli action.

What I am about to say is purely conjectural, but I believe that, when the cilia have condensed the mucus at the ostium and the mucus has formed an air seal in the ostium, as the cilia further attempt to push it into the nasal air passage there is produced a slight diminution of air pressure in the sinus which serves as a stimulus for a sneeze or a nose blow.

If the ostium happens to be in a pressure area of the residual air space, air will bubble through the mucus into the sinus and re-establish equalization of pressure. If, on the other hand, the ostium is located in an area of diminished pressure, the mucus is forced out into the nasal air passage by the air in the sinus until the bubble of mucus breaks. Thus, on the completion of the blow, atmospheric pressure is re-established in the sinus. So, no matter whether the sinus

ostia are located in areas of pressure or diminished pressure, nose blowing or sneezing tends to produce equalization of pressure in the sinuses.

CONCLUSIONS.

In this paper we have discussed the various factors which influence Bernoulli action in drainage of the accessory sinuses of the nose. We have seen that all of these factors are variable and this very variability explains why the productiveness of a given nose blow is unpredictable.

All of the favorable factors necessary for effective Bernoulli action are likely to be present in a moderately congested nose, and it is only on occasions of nasal and sinus congestion that such action is desirable. There is no necessity for draining a sinus which is empty.

Of all the factors which influence Bernoulli action, only one can be readily controlled voluntarily: that is the timing and force which one employs in blowing his nose.

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Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

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Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

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Manufacturer: Sonotone Corp., Elmsford, N. Y.

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Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

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Place: Army and Navy Club, Washington, D. C.

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Meeting: Palmer House, Chicago, Ill., Oct. 17, 1952.

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Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: January, 1954, Mexico City.

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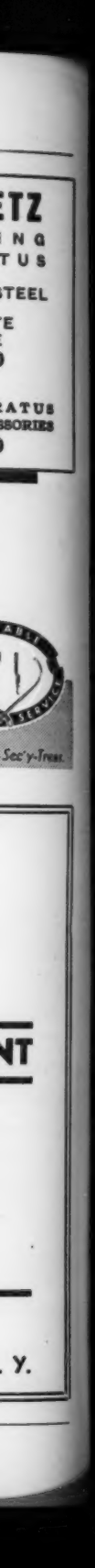

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